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An International System of Nomenclature for *Puccinia graminis* f. sp. *tritici*

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ABSTRACT

A new set of international differential hosts for characterizing the virulence of cultures of *Puccinia graminis* f. sp. *tritici* including the host resistance genes *Sr5*, *6*, *7b*, *8a*, *9b*, *9e*, *9g*, *11*, *17*, *21*, *30*, and *36* is proposed. Additionally, a resistant series that consists of host genes *Sr 13*, *22*, *24*, *25*, *26*, *27*, *31*, *32*, *33*, and *37* that have generally been resistant worldwide is recommended for evaluating bulk collections or composites of individual cultures. The use of additional differential hosts of local interest, especially those related to national breeding programs, is encouraged. Races are to be designated by a three-letter code (*Pgt*-code) followed by a hyphen and a listing of those host genes in the 'resistant' set on which the race was virulent. Data on local supplemental series should be separated from the *Pgt*-code by a slash. A central type-culture collection is proposed and will be maintained at the Cereal Disease Laboratory at St. Paul, MN, for use in breeding and research programs. Type-cultures will be evaluated against the resistance conferred by the known single genes.

The first key to what are now designated physiologic races of *Puccinia graminis* (Pers.) f. sp. *tritici* was published by Stakman and Levine (20) in 1922. This work followed the discovery of races 1 and 2 by Stakman and Pierneisel (23) in 1916. In 1918, race 3 was described by Levine and Stakman (7) and race 4 by Melchers and Parker (11). The physiologic race key of 1922 was based on the 12 differential host cultivars Little Club, Marquis, Kanred (later replaced by Reliance), Kota, Arnautka, Mindum, Speltz, Marz (known now as Spelmar), Kubanka, Acme, Einkorn, White Spring Emmer (now called Vernal), and Khapli (Table 2). Infection types (Table 1) were divided into groups, with 0, 1, and 2 indicating host resistance and infection types 3 and 4 indicating susceptibility. The X infection type was considered separately as a heterogeneous host response. Physiologic races were determined by a dichotomous key of resistant and susceptible host responses with the heterogeneous responses in a separate section of the key. The key was revised several times (21, 22, 24) and was last issued in 1962. These keys reflect gradual changes in the understanding of the pathogen-host relationship. The authors of the first key indicated that other hosts might be useful; however, this message was generally overlooked for nearly 30 years in the case of *P. g. f. sp. tritici*. The last of the series of keys using the Stakman differential set included a set of supplementary differential hosts for use with North American cultures. The differential hosts selected by Stakman and co-workers in 1916 were used worldwide until the mid-1960s, and they continue to be used to some extent in 1987. The changes in the classification of *P. g. f. sp. tritici* resulted from two major scientific events. The first was the development of the gene-for-gene concept, principally by Flor (4). Second, and as a consequence of the first, the development of host genotypes differing primarily by a single effective gene for resistance. The earliest lines that contained single genes for resistance to wheat stem rust were developed by Knott and Anderson (6), Watson and Luig (26), and Loegering and Harmon (8). A footnote in the 1962 key (24) for races of *P. g. f. sp. tritici* suggested that host lines with single genes might eventually lead to simplified and standardized procedures.

Table 1. Description of infection types used in physiologic specialization studies.

Infection Type	Symptoms	
0	Low	No uredia or other macroscopic sign of infection
;	Low	No uredia, but hypersensitive necrotic or chlorotic flecks of varying size present
1	Low	Small uredia often surrounded by necrosis
2	Low	Small to medium uredia often surrounded by chlorosis or necrosis
X	Low	Random distribution of variable-sized uredia on single leaf with a pure culture
Y	Low	Ordered distribution of variable-sized uredia, with larger uredia at leaf tip
Z	Low	Ordered distribution of variable-sized uredia, with larger uredia at leaf base
3	High	Medium-sized uredia that may be associated with chlorosis or rarely necrosis
4	High	Large uredia without chlorosis or necrosis

The infection types are often refined by modifying characters as follows;=, uredia at the lower size limit for the infection type;- , uredia somewhat smaller than normal for the infection type;+ , uredia somewhat larger than normal for the infection type;+ , uredia at the upper size limit for the infection type;C, more chlorosis than normal for the infection type;and N, more necrosis than normal for the infection type. Discrete infection types on a single leaf when infected with a single biotype are separated by a comma (e.g., 4,; or 2=,2+ or 1,3C). A range of variation between infection types is recorded by indicating the range, with the most prevalent infection type listed first (e.g., 23 or ; 1C or 31N). Infection types 2, X, Y, Z and 3 are often considered inadequate resistance levels for use in commercial cultivars.

Table 2. Genotype of the hosts used in the Stakman series of differential hosts for *Puccinia graminis* f. sp. *tritici* as currently understood

Differential Hosts ^a	Sr gene(s)	Remarks ^b
Little Club	LC	Generally ineffective
Marquis	7b,18,19,20,X	SrX is important in N. America
Reliance	5,16,18,20	Sr5 is included in thePgt-set
Kota	7b,18,19,28,Kt'2'	Sr28 is important in Indian subcontinent
Arnuatka	9d,a	Differential in N. America
Mindum	9d,a,b	Differential in N. America
Spelmar	9d,a,b	Differential in N. America
Kubanka	9g,c	Src detected in N. America
Acme	9g,d	Srd detected in N. America
Einkorn	21	Sr21 is included in Pgt-set
Vernal	9e	Sr9e is included in Pgt-set
Khapli	7a,13,14	Sr13 is an important resistance

^a Stakman et al (24).

^b SrLC, 14, 16, 18, 19, 20, and Kt'2' are generally ineffective worldwide, except in sexually reproducing populations and against *P. graminis* f. sp. other than *tritici*. Sra, b, c, d are postulated to be present based on host response and are effective to some North America cultures. Sr7a results in a low infection type that is too unstable to use with current technology.

The acceptance of single-gene differential hosts for race identification came rapidly, involving different systems of nomenclature, i.e., Australia in 1963 (26), Canada in 1965 (6), and the United States in 1972 (17). Generally, other laboratories have used one of these three systems for classifying races. Separate systems were developed in India (1, 19). The change to single-gene differential hosts was useful in breeding resistant cultivars and in understanding the changes in the pathogen population (13). However, the characterization of virulence of the pathogen populations on a global basis was fraught with difficulties caused by other resistance genes in the host background into which the single genes had been transferred, and to differences in hosts and nomenclature systems used. Efforts to correct this were proposed or attempted by several workers (10, 15, 25).

This paper establishes an international system of nomenclature for *P. g. f. sp. tritici* that should facilitate worldwide communication between breeders and pathologists; the breeding of resistant wheat cultivars; and local and global monitoring of evolution of virulence in the pathogen. It also establishes a type culture collection in support of these objectives. The system permits an evaluation of cultures in the area of origin and a complete phenotype description of the type cultures submitted to the Cereal Disease Laboratory.

MATERIALS AND METHODS

This system comprises: 1) a local phenotypic evaluation of samples from the pathogen population on 12 differential hosts each with a single gene resistance; 2) a local evaluation on 12 resistant lines each with a single gene; 3) an evaluation on a locally selected supplemental series; and 4) testing of type cultures on a comprehensive set of host lines with single gene resistances and the Stakman differential hosts (Table 2) under controlled conditions at the Cereal Disease Laboratory, St. Paul, MN. For purposes of race designation, evaluation will be done on seedling plant leaves.

Based on previous experience (10, 13, 19), 12 single-gene host lines (Table 3) with a differential response to a significant portion of the worldwide pathogen population were selected as potential differential hosts. Additional criteria for selection were a recognizable low infection type and stability of the low infection type over the range of environmental conditions found in most research laboratories. A low infection type in a gene-for-gene system is defined as any infection type that is detectably less than the fully compatible infection type (Table 1). Differential hosts were selected so that the low infection type would normally be less than a 23. From this group of potential differential hosts, those that were important in the Stakman Series (Table 2) and in the other differential sets (1, 3, 6, 19, 26) were retained to maintain historical continuity. Additionally, genes that were effective in commercial cultivars were also emphasized.

Table 3. Possible hosts with the required single genes for resistance to *Puccinia graminis* f.sp. *tritici* for the Pgt differential set.

Sr gene	Single-gene line	Winter habit cultivars	Spring habit cultivars
5	ISr5-Ra	Cheyenne	Summit
21	triticium monococom derivative		Einkorn
9e	Verstein		Vernal
7b	ISr7b-Ra	Hart	Red Fife
11	ISr11-Ra		Gabo ^a
6	ISr6-Ra		McMurachy
8a	ISr8-Ra	Flavio	Mentana
9g	CnSSr9g		Kubanka
36(Tt-1)	W2691SrTt-1	Kenosha	Idaed 59
9b	W2691Sr9b		Gamenya
30	BtSr30Wst		Festiguay
17	Combination VII	Scout 66 ^b	Regent ^c

^a Additional gene for resistance to some North American cultures.

^b Sr9d also present

^c Sr7b and 9d also present

Evaluation for resistance on a worldwide basis has shown a number of lines with single-gene resistance to be almost universally effective (9,13,17) against the *P. graminis* populations. Because of the value of these genes to wheat breeders and the importance of detecting evolving virulence toward them, we propose that lines with resistance of this type comprise the resistance series (Tables 4 and 5). This series can be tested most efficiently by inoculation with bulk collections (12) until virulence is detected. After virulence is detected, that host resistance should be evaluated to individual cultures. For the previously given reasons there are advantages for using adapted commercial cultivars that possess these genes singly, rather than attempting to use a near-isogenic series in which the recurrent parent may be poorly adapted to some environments (Table 4). However, caution in interpretation of infection type data is required because resistance in a line may be caused by a second, previously undetected, host gene.

Supplemental differentials also need to be selected to characterize the pathogen population relative to the resistance in local cultivars and to the host genotypes in the local breeding program. To permit the important task of evaluating hosts of local interest, the number of international differential hosts was kept at a minimum.

Type cultures for each race from each country where rust studies are done can be sent to the Cereal Disease Laboratory at St. Paul, MN. Type cultures will be evaluated to all the known single genes for resistance and to the Stakman international differential series. This provides for a more complete

phenotypic description of the virulence/ avirulence of the pathogen and ensures an important historical continuity.

Type cultures will be stored at -196 C so that they are available for future studies. The type cultures will be generally evaluated under a standard set of environmental conditions, i.e., 18 C and more than 10,000 lx of light for a 16-hr day. However, resistance genes known to be effective only under other environmental conditions will be evaluated separately. The results obtained may vary with those obtained at the originating laboratory; however, both are correct phenotypic descriptions for the environment evaluated. The results of the testing of type cultures will be made available annually in a suitable international forum. A modified system of nomenclature was chosen from the available systems (12). Differential hosts are placed in sets of four, and a letter is assigned to each of the 16 possible (24) combinations of infections types (Table 6) that are possible. Sets of four minimize the length of the code with enough characters to describe the codes. The letters B through T minus vowels are used for race designation. New subsets of differential hosts can be added to the right without affecting the previously used coding with the letter codes for new subsets added to the right.

Table 4. 'Resistant' set recommended for supplementing the Pgt differential set for *Puccinia graminis* f. sp. *tritici*.

Wheat lines or cultivars			
Sr gene	Single gene	Winter habit	Spring Habit
13	W2691Sr13		Wialki
22	SwSr22T.b.		
24	BtSr24Ag	Agent	
25	LCSr25Ars	Agrus	Agatha ^a
26	Line U		Kite
27	W2691Sr27		
29	Pusa*4/Etiole de Choisy	Etiole de Choisy	
31	W3498Sr31Kvz	Clement	
32	ER 5155 ^b		
33	RL 5405		
37(Tt-2)	W2691Sr37T.t.		

^a Sr5, 9g, 16 may also be present.

^b Sr9f may also be present.

RESULTS

The infection types produced on the host of the *Pgt* differential set by selected races of the pathogen population from North America, Australia, and other areas (18) are shown in Tables 6-12. It is important to recognize that this differential set does not provide a complete phenotypic description of the pathogen virulence, since only selected host resistance genes are evaluated. Thus, we know that race MCC (Table 6) can be subdivided on SrTmp.

The mean ranges of low infection types produced with the *Pgt* differential set are shown in Table 13. These infection types are not included in Tables 6-12, because for the purpose of international race

classification, it is proposed that all low infection types be treated as equal. This avoids subdividing races on the basis of small environmental differences rather than genetic differences in the pathogen. This procedure may lump cultures of slightly different genotype together. In special studies of type cultures, these differences can be described.

The host lines are arranged in subsets of four with the historical differentials to the left; and within subsets host-pathogen interactions known to be more difficult to classify are to the right.

The first four differential hosts of the new *Pgt* set represent genes found and considered to be of major importance in the Stakman series. The relationship between the Stakman race numbers and the first letter in the Pgt-designation is not always possible to determine as some of the genes represented in the Stakman Series (Table 2) are not included. The genes known to be missing are *SrLC* (Little Club), *SrX*, *18*, *19*, *20* (Marquis), *Sr16*, *18*, *20* (Reliance), *Sr18*, *28*, Kt'2' (Kota), *Sr9d*, and several genes conditioning an X infection type in Arnautka, Mindum, and Spelmar, several genes conditioning an X infection type in Kubanka and Acme, and *Sr7a*, *13*, and *14* (Khapli). The Stakman races *11*, *15*, *17*, *21*, *34*, *40*, and *56*, which have been historically important in some wheat growing areas, are unaffected by these exclusions. Conversely, races *14*, *29*, *32*, *98*, *113*, *117*, *126*, *134*, and *151* are affected.

The mean low infection types produced with lines in the resistant series (Table 4) are shown in Table 5. These tests were generally conducted at 18 C with a 16-hr day with over 10,000 lx of fluorescent light supplemented with sunlight.

Table 5. Expected low infection types for the 'resistant' set recommended for supplementing the Pgt differential set for *Puccinia graminis* f. sp. *tritici*.

Sr gene	Expected low infection type ^a	Remarks	Source
13	2+	Most effective above 25 C	<i>Triticum durum</i>
22	2=	Not currently used	<i>T. Monococcum</i>
24	2+-	Used in N and S. America and South Africa	<i>Agropyron elongatum</i>
25	2-	Juvenile resistance	<i>A. elongatum</i>
26	;2-	Used in Australia	<i>A. elongatum</i>
27	0;2-	Used in rye and triticale	<i>Secale cereale</i> 'Imperial'
29	2+-	Used in Europe	'Etiole de Choisy'
31	;2-	Used worldwide	<i>S. cereale</i> 'Petkus'
32	;2-	Not currently used	<i>T. speltoides</i>
33	;2	Not currently used	<i>T. squarrosa</i>
37(Tt-2)	0;	Frequently off-type plants	<i>T. timopheevi</i>

^aExpected low infection type for the recommended backgrounds (Table 4). Some variation may occur depending on the entire host or pathogen genotype, as well as with changes in the environment. Data in part from Roelfs and McVey(17) and Kuig(9).

DISCUSSION

The identification of virulence phenotypes has been a very important part of the program to breed resistant host cultivars as well as in studying regional disease spread and the evolution of virulence in the pathogen (12). Nevertheless, the naming of phenotypic variants has come under criticism, such as for arbitrariness, number of possible phenotypes, and the problem of adding new differential hosts (2). The underlying stability in the *P. g. f. sp. tritici-Triticum* spp. host parasite system is such that naming these phenotypic combinations has been and will continue to be useful (13). Sexually reproducing populations are more heterogeneous and many more avirulences exist that are not detected by the *Pgt*-system (14).

The evolutionary changes that occur in a pathogen population such as *P. g. f. sp. tritici* will necessitate changes in differential hosts, to maintain relevancy of race determination. Experience has indicated that a review of the system used is required at least every 10 yr. No system can be expected to serve indefinitely. However, for purposes of international communication, the changes should be infrequent and made only when they are significant. Changes in pathogen virulence are often related to the resistance present in the host population. Thus, those genes currently occurring in commercial cultivars and those now being introduced into breeding programs were placed in the 'resistant' set. This system should serve for at least 10 years as the development of cultivars takes more than 8 yr. Thus, the selection pressure that will be exerted on the pathogen populations has been anticipated to some extent. The system is designed so that a race designation can be assigned to virulence combinations directly, eliminating the need to contact a central source before publishing data. It is hoped that local differential hosts will be added. In parts of the world, some of the genes represent in the proposed differential series always confer resistance or susceptibility; such hosts lines probably need only be evaluated with representative samples of the pathogen population annually rather than be included in all race characterizations. An example would be the line with *Sr9g* in North and South America, where avirulence is unknown in the asexual population after the evaluation of more than 20,000 isolates during the past 29 yr.

It is proposed that the international race designation be reported with *Pgt* notation, followed by a slash (/), the *Sr* genes in the 'resistance' series on which the culture is virulent followed by the information on the supplemental series. If no lines in the resistance set are susceptible indicate as *Pgt*-TPM-SrO/; if the set was not tested use *Pgt*-TPM-Sr-/ . This permits anyone to derive the virulence/ avirulence formula for the *Pgt* set quickly. In reports using only a few cultures the authors may choose to use a virulence/ avirulence formula, although we suggest giving the *Pgt* race in addition.

The differential host genes were selected on the following basis: a) stability and ease of classification under the range of environmental conditions generally available for testing at most laboratories, b) both low and high infection types can be detected in most areas of the world, and c) the phenotypic difference detected is important in breeding for resistance, or in studies of evolution and epidemiology. Additionally, we attempted to maintain a connection with the Stakman series. A type culture is proposed for comparative studies and to facilitate international access for scientific purposes. Type cultures will be evaluated under a specified set of conditions on all known single sources of resistance, as well as to the Stakman differential series. The data will be returned to the originating laboratory and also published annually in a readily accessible publication. As the system becomes established, less frequent publication may be needed depending on the number of type cultures submitted. Samples of each type culture will be stored at the Cereal Disease Laboratory and will be available for future use. It is proposed that, where possible, the laboratory of origin should also store the culture, because field evaluation can only be done at that location.

The background genotype for producing single-gene host lines by backcrossing was generally selected for susceptibility to the local population of *P. graminis* f. sp. *tritici* and local adaptation and often proved to

have undetected genes for resistance when evaluated elsewhere. Because of differences in environmental conditions among locations, these lines often grow poorly and are sometimes very susceptible to other diseases, making maintenance of seed stocks in some environments difficult. These problems may be partly avoided by using adapted cultivars that possess the selected resistance gene, but caution must be used, to assure that the desired gene pair is responsible for the low infection type expressed (Table 13). It is known, for example, that some cultures from the Indian subcontinent are avirulent on a resistance gene other than Sr6, 9g, or U in the ISr6-Ra line used as a differential host (Roelfs, unpublished). This population must be evaluated on the W2691Sr6 line to evaluate the Sr6 response.

Seed of the Pgt differential and 'resistant' sets will be available in small quantities from the Cereal Disease Laboratory. The long-range goal is to have single-gene host lines available for each of the differential and resistant host lines in an isoline and in spring and winter habit genotypes without other major genes for resistance to *P. g. f. sp. tritici*. It is hoped that such cultivars will be more useful in many areas of the world where a lack of adaptation to environmental and local pests have frequently prevented the use or seed increase of the current isolines.

Table 6. Pgt-code for the 12 Pgt differential hosts for *Puccinia graminis* f. sp. *tritici* in ordered subsets of four

Pgt Code	Subset ^a	Infection type produced on host lines with Sr				
	1	5	21	9e	7b	
	2	11	6	8a	9g	
	3	36	9b	30	17	
B		Low	Low	Low	Low	Low
C		Low	Low	Low	Low	High
D		Low	Low	High	High	Low
F		Low	Low	High	High	High
G		Low	High	Low	Low	Low
H		Low	High	Low	Low	High
J		Low	High	High	High	Low
K		Low	High	High	High	High
L		High	Low	Low	Low	Low
M		High	Low	Low	Low	High
N		High	Low	High	High	Low
P		High	Low	High	High	High
Q		High	High	Low	Low	Low
R		High	High	Low	Low	High
S		High	High	High	High	Low
T		High	High	High	High	High

^a Pgt-code consists of the designation for subset 1 followed by that for subset 2, etc. For example, race TTT is virulent (high infection type) on all 12 differential hosts and race DCL is virulent on differential hosts with Sr9e, 9g, and 36. Low and high infection types indicate an incompatible and a compatible host-pathogen interaction, respectively.

LITERATURE CITED

1. Bahadur, P., Nagarajan, S., and Nayar, S. K. 1985. A proposed system for virulence designation in India. Proc. Indian Acad. Sci. 95:29-34.
2. Caten, C. E., 1987. The concept of race in plant pathology. Pages 21-37 in: Populations of Plant Pathogens: Their Dynamics and Genetics. Blackwell Scientific Publications, Oxford.
3. Coelho, E. T., Silva, A. R., and Barcellos, A. L. 1971. Levantamento de racas fisiologicas de *Puccinia graminis tritici* no Brasil. Pesqui. Agropecu. Bras, Ser. Agron. 6:115-125.
4. Flor, H. H. 1971. Current status of the gene-for-gene concept. Annu. Rev. Phytopathol. 9:275-296.
5. Green, G. J. 1965. Stem rust of wheat, rye and barley in Canada in 1964. Can. Plant Dis. Surv. 45:23-29.
6. Knott, D. R., and Anderson, R. G. 1965. The inheritance of stem rust resistance, I. The inheritance of stem rust resistance in ten varieties of common wheat. Can. J. Agric. Sci. 36:174-195.
7. Levine, M. N., and Stakman, E. C. 1918. A third biologic form of *Puccinia graminis* on wheat. J. Agric. Res. 13:174-195.
8. Loegering, W. Q., and Harmon, D. L. 1969. Wheat lines near-isogenic for reaction of *Puccinia graminis tritici*. Phytopathology 59:456-459.
9. Luig, N. H. 1983. A Survey of Virulence Genes in Wheat Stem Rust, *Puccinia graminis* f. sp. tritici. Verlag Paul Parey, Berlin. 199 pp.
10. Luig, N. H., and Latter, B. D. IT. 1983. A new international classification system for *Puccinia graminis tritici*. Page 138 in: Proc. 4th Int. Congr. Plant Pathol. Melbourne, Australia.
11. Melchers, L. E., and Parker, J. H. 1918. Another strain of *Puccinia graminis*. Kans. Agric. Exp. Stn. Circ. 68. 4 pp.
12. Roelfs, A. P. 1984. Race specificity and methods of study. Pages 132-164 in: The Cereal Rusts, Vol. 1. Origins, Specificity, Structure, and Physiology. W. R. Bushnell and A. P. Roelfs, eds. Academic Press, Orlando, FL. 546 pp.
13. Roelfs, A. P. 1984. Wheat and rye stem rust. Pages 3-37 in: The Cereal Rusts, Vol. 11. Diseases, Distribution, Epidemiology and Control. A. P. Roelfs and W. R. Bushnell, eds. Academic Press, Orlando, FL. 606 pp.
14. Roelfs, A. P., and Groth, J. V. 1980. A comparison of virulence phenotypes in wheat stem rust populations reproducing sexually and asexually. Phytopathology 70:855-862.
15. Roelfs, A. P., and Martens, J. W. 1984. Proposal for an international system of race nomenclature for *Puccinia graminis* f. sp. tritici. Pages 99-102 in: Proc. 6th Eur. Mediter. Cereal Rusts Conf., Grignon, France.

16. Roelfs, A. P., and McVey, D. V. 1973. Races of *Puccinia graminis* f. sp. *tritici* in the USA during 1972. *Plant Dis. Rep.* 57:880-884.
17. Roelfs, A. P., and McVey, D. V. 1979. Low infection types produced by *Puccinia graminis* f. sp. *tritici* and wheat lines with designated genes for resistance. *Phytopathology* 69:722-730.
18. Saari, E. E., and Prescott, J. M. 1985. World. distribution in relation to economic losses. Pages 259-298 in: *The Cereal Rusts, Vol. 11. Diseases, Distribution, Epidemiology and Control.* A. P. Roelfs and W. R. Bushnell, eds. Academic Press, Orlando, FL. 606 pp.
19. Sawhney, R. N., and Goel, L. B. 1983. New differential testers for the characterization of Indian races of the wheat stem rust and leaf rust pathogens for virulence. *Indian Phytopathol.* 36:3 12-32 1.
20. Stakman, E. C., and Levine, M. N. 1922. The determination of biologic forms of *Puccinia graminis* on *Triticum* spp. *Minn. Agric. Exp. Stn. Tech. Bull.* 8 pp.
21. Stakman, E. C., and Levine, M. N. 1938. Analytical key for the identification of physiologic races of *Puccinia graminis tritici*. *Div. of Cereal Crops and Dis. U. S. Dept. Agric. and Minn. Agric. Exp. Stn.* 7 pp.
22. Stakman, E. C., Levine, M. N., and Loegering, W. Q. 1944. Identification of physiologic races of *Puccinia graminis tritici*. *U.S. Dept. Agric. Bur. Entomol. Plant Quarant.* E-617. 27 pp.
23. Stakman, E. C., and Piemeisel, F. J. 1917. A new strain of *Puccinia graminis*. (Abstr.) *Phytopathology* 7:73.
24. Stakman, E. C., Stewart, D. M., and Loegering, W. Q. 1962. Identification of physiologic races of *Puccinia graminis* var. *tritici*. *U.S. Dept. Agric., Agric. Res. Serv.* E-617. 53 pp.
25. Watson, I. A. 1977. The national wheat rust control programme in Australia. *Vestn. S-kh. Nauki* 6:123-133.
26. Watson, I. A., and Luig, N. H. 1963. The classification of *Puccinia graminis* var. *tritici* in relation to breeding resistant varieties. *Proc. Linn. Soc. N. S. W.* 88:235-258.