# Pathogenicity Associations in Puccinia recondita tritici

L. E. Browder and M. G. Eversmeyer

Research Plant Pathologists, Agricultural Research Service, U.S. Department of Agriculture, Plant Pathology Department, Kansas State University, Manhattan, KS 66506.

Cooperative investigations of the Agricultural Research Service, U.S. Department of Agriculture and the Kansas Agricultural Experiment Station, Department of Plant Pathology; Contribution No. 644, Kansas Agricultural Experiment Station, Manhattan.

Accepted for publication 13 December 1976.

ABSTRACT

BROWDER, L. E., and M. G. EVERSMEYER. 1977. Pathogenicity associations in Puccinia recondita tritici. Phytopathology 67: 766-771.

Pathogenicity of population samples of *Puccinia recondita* f. sp. *tritici* from eight geographic regions of the United States was analyzed by calculating virulence frequencies to single lines and by association of pathogenicity and association of virulence to all possible combinations of two lines within a set of nine wheat lines, each with a different, single, known gene for low reaction to *P. recondita*. Associations of pathogenicity were studied by calculating Pathogenicity Association Coefficients (PAC) and Virulence Association Coefficients (VAC) for each possible pair of lines. Marked differences were found in both virulence frequencies and in associations of pathogenicity in different geographic regions. Combinations of host genes for low reaction which can be effectively used for control of leaf rust on wheat are characterized by high PAC and low VAC. These coefficients also are useful in detecting gene combinations in wheat cultivars grown during the time the parasite population sample was collected.

*Additional key words*: wheat leaf rust, disease resistance, host:parasite relationships.

The pathogenicity of *Puccinia recondita* Rob. ex Desm. sp. *tritici* classically has been described by the identification and reporting of pathogenic races represented in collections from populations (3). An International Standard set of differential cultivars was specified (15) and used (14) until investigators became aware that it was not adequate for describing pathogenicity when the information was used to guide wheat breeding programs (1, 23). At least three different sets of supplemental differentials have been specified (2, 23, 25) and three nomenclatural methods proposed (1, 17, 23) to improve the usefulness of data obtained from *P. recondita* pathogenicity surveys. Three other methods of nomenclature for pathogenic races in the other cereal rust fungi have been proposed (12, 13, 20).

Each of the above methods used a taxonomic approach. Each of the methods provides a "name" for any possible variant within the experimental materials used. Pathogenic race names derived by these methods are either inadequate to describe the variation in *P. recondita* populations or are unwieldy in communication. The variation existing in the *Triticum aestivum* L. em. Thell.:*P. recondita* system is so extensive (18) that it would be impossible to describe and name all the races if all the existing variation was included in the taxonomic system. Kernkamp (16) has suggested that "efforts to do so may be purely academic gymnastics." Day (8) suggested that further development of studies of pathogenic specialization "will no doubt be in the direction of regarding race designations as largely unnecessary" and that "the most important information is the frequency of certain critical virulence genes in the pathogen population." He also suggested that "combinations of virulence genes" may be important. These ideas suggest that a population genetics approach to the study of pathogenic specialization may be more useful than a taxonomic approach. The major objective of pathogenicity surveys should be to adequately describe pathogen populations so that the information can be used effectively in breeding programs rather than attempting to name all the variants present in the fungal population.

J. F. Schafer (*personal communication*) proposed a paired-comparison analysis for survey data in which pathogenicity (avirulence or virulence) to paired lines of unknown reaction genotype were compared in a $2 \times 2$ chi-square test for independence. He suggested that significant chi-square values, indicating nonrandom occurrence of pathogencity to the lines, was evidence of similarity of reaction genotype in the two lines. He has shown several probable genetic relationships of differentials used in pathogenicity surveys. This method also has been used to show a similarity between a host line of previously unknown genotype and a near-isogenic line having *Lrll* (4). The model on which this analysis is based (3) is symmetrical. Thus, the analysis can be applied to either side of the host:parasite interaction.

We have used virulence frequencies to host lines having a single known gene and a modification of Schafer's analysis, without the chi-square test, to portray variation in pathogenicity of *P. recondita* to paired lines of known reaction genotype. We present these analyses in this paper.

## MATERIALS AND METHODS

We used infection-type data from nine lines of wheat, *T. aestivum*, each having a different, single, known gene

for low reaction to *P. recondita* (9, 10, 11), and each inoculated with each of 809 single-pustule isolates of *P. recondita*. These isolates were made from 429 cultures obtained from 653 collections received in 1972 from 34 wheat-growing states. The nine lines were: LR1(TC), RL6003; LR2A(TC), RL6000; LR2D(PL), RL6001; LR3(TC), RL6002, LR10(TC), RL6004; LR16(TC), RL6005; LR17(TC), RL6008; LR18(TC), RL6009; LR3B(TC), RL6007. The term pathogenicity as used in this paper means the capacity to produce disease. Virulence is used to mean the capacity to produce severe disease; avirulence means the inability to produce severe disease. Each of the terms is used in a specific sense, in relation to a specific host genotype. Isolation and inoculation methods used in this paper were previously described (3). The infected host lines were placed at 20 ± 2 C in a plant growth chamber with a 12 hr day at about 21,520 lux. Infection types were observed 9-10 days later and coded by a system in which amount of sporulation, lesion size, and unique lesion characters were coded independently (7). Values of 8 or 9 in the sporulation amount code were considered to indicate virulence and values of 7 or less were considered to indicate virulence of specific isolates to specific lines. Thus, the infection-type data were interpreted as, or transformed to, pathogenicity data. The pathogenicity data were then sorted as to eight geographic regions of the United States from which the collection it represented had been made.

Virulence frequency to each of the nine lines in each of the eight geographic areas was calculated by dividing the number of virulent isolates by the total number of isolates in each sample.

When pathogenicity of each isolate to each host line is classified as avirulent or virulent, pathogenicity of that isolate to any pair of host lines, A and B, can be classified as: $\text{Avirulent}_A$:$\text{Avirulent}_B$ ($A_A$:$A_B$); $\text{Avirulent}_A$:$\text{Virulent}_B$ ($A_A$:$V_B$); $\text{Virulent}_A$:$\text{Avirulent}_B$ ($V_A$:$A_B$); or $\text{Virulent}_A$:$\text{Virulent}_B$ ($V_A$:$V_B$). All isolates represented in the data were classified in this way with respect to all possible combinations of two lines in the set of nine lines used. For example, an isolate avirulent to both LR1(TC) and LR10(TC) would be classified as $A_1$:$A_{10}$. Pathogenicity Association Coefficients (PAC) and Virulence Association Coefficients (VAC) were calculated in a similar way as coefficients of resemblence in numerical taxonomy (22), as:

$$PAC_{A:B} = \frac{(\text{No. of isolates } A_A{:}A_B) + (\text{No. of isolates } V_A{:}V_B)}{\text{Total No. of isolates in sample}},$$

and

$$VAC_{A:B} = \frac{\text{No. of isolates } V_A{:}V_B}{\text{Total No. of isolates in sample}}$$

A PAC value and a VAC value were calculated for each possible host-line pair in each of the eight geographic-region samples by this method. The necessary sorting, tabulation, and calculations were done by automatic data processing equipment (6).

## RESULTS

Virulence frequency data for the nine lines in each of the eight regions are shown in Table 1. These data showed large differences in virulence frequencies to certain lines from region to region, but similar virulence frequencies to other lines in all regions. For example, virulence to LR1(TC), having *Lr1*, was 12-18% in the central regions, but was 83% in the southeastern United States. This difference in frequency of virulence to LR1(TC) is at least partly due to the requirement of that virulence in *P. recondita* to attack Blueboy (C.I. 14031) (5) in the southeastern region where Blueboy was widely grown, and where a severe epidemic of wheat leaf rust occurred in 1972 (19). No cultivar currently grown in the central area has *Lr1*, but that gene for low reaction does occur in advanced lines in hard red winter wheat breeding programs (Browder and Eversmeyer, *unpublished*), and it probably will be deployed in the central United States in the future.

Frequency of virulence to LR3(TC) was very high (93-97%) in the central regions but was only 70% in the

TABLE 1. Virulence frequencies of *Puccinia recondita* f. sp. *tritici* to nine lines of *Triticum aestivum* in samples from eight geographic regions of the United States in 1972

| | No. isolates (N) in sample and % frequency virulence per region[a]: | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | S.C. | Central | N.C. | S.E. | N.E. | N. Eng. | West | N.W. |
| Near-isogenic wheat line: | N=117 | N=129 | N=147 | N=163 | N=167 | N=9 | N=7 | N=21 |
| LR1(TC) | 17 | 12 | 18 | 83 | 68 | 67 | 86 | 19 |
| LR2A(TC) | 7 | 5 | 12 | 51 | 19 | 67 | 0 | 19 |
| LR2D(PL) | 13 | 12 | 16 | 56 | 38 | 89 | 86 | 19 |
| LR3(TC) | 97 | 96 | 93 | 70 | 84 | 33 | 14 | 95 |
| LR10(TC) | 71 | 55 | 69 | 73 | 80 | 100 | 86 | 67 |
| LR16(TC) | 3 | 5 | 5 | 4 | 5 | 22 | 0 | 5 |
| LR17(TC) | 6 | 10 | 9 | 32 | 29 | 78 | 71 | 0 |
| LR18(TC) | 7 | 0 | 5 | 6 | 2 | 11 | 0 | 5 |
| LR3B(TC) | 7 | 13 | 7 | 10 | 19 | 22 | 0 | 0 |

[a]S.C. (South Central) = Texas, Oklahoma, and New Mexico; Central = Kansas, Colorado, and Nebraska; N.C. (North Central) = South Dakota, North Dakota, Minnesota and Wisconsin; S.E. (Southeast) = Louisiana, Mississippi, Alabama, Georgia, Florida, Tennessee, Kentucky, South Carolina, North Carolina, and Virginia; N.E. (Northeast) = Arkansas, Missouri, Iowa, Indiana, Ohio, and Michigan; N. Eng. (New England) = New England States, Pennsylvania, Maryland, and West Virginia; N.W. (Northwest) = Montana, Idaho, and Wyoming; and West = California, Washington, Oregon, Nevada, Utah, and Arizona.

TABLE 2. Pathogenicity Association Coefficient (PAC)[a] and Virulence Association Coefficient (VAC)[b] data of *Puccinia recondita* f. sp. *tritici* to 11 of the 36 possible combinations of near-isogenic lines of *Triticum aestivum* estimated in samples from eight geographic regions of the United States in 1972

| | | No. isolates (N) in sample, PAC and VAC per region[c]: | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | S.C. | | Central | | N.C. | | S.E. | | N.E. | | N. Eng. | | West | | N.W. | |
| | Comparison | N=117 | | N=129 | | N=147 | | N=163 | | N=167 | | N=9 | | N=7 | | N=21 | |
| Item | of lines: | PAC | VAC | PAC | VAC | PAC | VAC | PAC | VAC | PAC | VAC | PAC | VAC | PAC | VAC | PAC | VAC |
| 1 | LR1(TC):LR2A(TC) | 0.88 | 0.06 | 0.93 | 0.05 | 0.88 | 0.10 | 0.67 | 0.51 | 0.50 | 0.19 | 1.00 | 0.67 | 0.14 | 0.0 | 0.90 | 0.14 |
| 2 | LR1(TC):LR2D(PL) | 0.92 | 0.11 | 0.93 | 0.09 | 0.90 | 0.12 | 0.64 | 0.52 | 0.62 | 0.34 | 0.78 | 0.68 | 1.00 | 0.86 | 0.90 | 0.14 |
| 3 | LR1(TC):LR3(TC) | 0.17 | 0.16 | 0.10 | 0.09 | 0.12 | 0.12 | 0.57 | 0.55 | 0.52 | 0.52 | 0.0 | 0.0 | 0.0 | 0.0 | 0.24 | 0.19 |
| 4 | LR1(TC):LR10(TC) | 0.28 | 0.08 | 0.50 | 0.09 | 0.36 | 0.12 | 0.68 | 0.62 | 0.77 | 0.62 | 0.67 | 0.67 | 1.00 | 0.86 | 0.24 | 0.05 |
| 5 | LR1(TC):LR18(TC) | 0.79 | 0.02 | 0.89 | 0.0 | 0.84 | 0.03 | 0.23 | 0.06 | 0.33 | 0.02 | 0.22 | 0.0 | 0.14 | 0.0 | 0.86 | 0.05 |
| 6 | LR1(TC):LR3B(TC) | 0.83 | 0.03 | 0.82 | 0.04 | 0.82 | 0.03 | 0.19 | 0.06 | 0.40 | 0.14 | 0.11 | 0.0 | 0.14 | 0.0 | 0.81 | 0.0 |
| 7 | LR3(TC):LR10(TC) | 0.71 | 0.69 | 0.51 | 0.51 | 0.65 | 0.64 | 0.48 | 0.45 | 0.65 | 0.64 | 0.33 | 0.33 | 0.0 | 0.0 | 0.62 | 0.62 |
| 8 | LR10(TC):LR16(TC) | 0.32 | 0.03 | 0.48 | 0.04 | 0.36 | 0.05 | 0.29 | 0.03 | 0.23 | 0.04 | 0.22 | 0.22 | 0.14 | 0.0 | 0.38 | 0.05 |
| 9 | LR16(TC):LR17(TC) | 0.92 | 0.0 | 0.88 | 0.01 | 0.86 | 0.0 | 0.65 | 0.01 | 0.67 | 0.0 | 0.22 | 0.11 | 0.29 | 0.0 | 0.95 | 0.0 |
| 10 | LR16(TC):LR18(TC) | 0.91 | 0.0 | 0.96 | 0.0 | 0.91 | 0.01 | 0.92 | 0.0 | 0.93 | 0.0 | 0.89 | 0.11 | 1.00 | 0.0 | 0.90 | 0.0 |
| 11 | LR17(TC):LR18(TC) | 0.87 | 0.0 | 0.91 | 0.0 | 0.87 | 0.0 | 0.62 | 0.0 | 0.69 | 0.0 | 0.33 | 0.11 | 0.29 | 0.0 | 0.95 | 0.0 |

[a] $PAC_{A:B} = \frac{A_A:A_B + V_A:V_B}{\text{Total no. of isolates}}$

[b] $VAC_{A:B} = \frac{V_A:V_B}{\text{Total no. of isolates}}$

[c] S.C. (South Central) = Texas, Oklahoma, and New Mexico; Central = Kansas, Colorado, and Nebraska; N.C. (North Central) = South Dakota, North Dakota, Minnesota and Wisconsin; S.E. (Southeast) = Louisiana, Mississippi, Alabama, Georgia, Florida, Tennessee, Kentucky, South Carolina, North Carolina, and Virginia; N.E. (Northeast) = Arkansas, Missouri, Iowa, Indiana, Ohio, and Michigan; N. Eng. (New England) = New England States, Pennsylvania, Maryland, and West Virginia; N.W. (Northwest) = Montana, Idaho, and Wyoming; and West = California, Washington, Oregon, Nevada, Utah, and Arizona.

southeastern region. The virulence frequency to LR3(TC) was low in the western and New England regions, but the number of isolates in these sampling regions was relatively small. The frequency of cultivars with *Lr3* that are currently grown in the three central regions is low, but it has been high in the past. Previously-grown cultivars Kawvale, Pawnee, Ponca, and Ottawa have *Lr3* (Browder and Eversmeyer, *unpublished*). Virulence to these cultivars increased [with an increase in Unified Numeration (UN) races 2 and 5 and a decline in UN race 9] during the time these cultivars were grown (14) and it has not yet declined. Virulence to LR3(TC) declined in the southeastern region (with a decrease in UN race 13 and an increase in UN race 9) in 1972. We cannot explain these shifts in either area. However, in the southeastern region, avirulence to LR3(TC) was associated with virulence to LR1(TC) and to LR10(TC). The frequency of $V_1$:$A_3$:$V_{10}$ was 29% of the 163 isolates from the southeastern region.

The virulence frequency to LR10(TC) was high in all regions, and the virulence frequency to LR16(TC), LR18(TC), or LR3B(TC) was relatively low in all regions. Virulence frequencies to each of the nine host lines were similar in the south-central, central, and north-central regions. This indicates that these regions form one epidemiologic area for wheat leaf rust.

The complete PAC and VAC data obtained from the pathogenicity association analysis consisted of 36 PAC and 36 VAC for each of the eight regions, or 576 pieces of data, and were too unwieldy to present in published form. Thus, we present data for only 11 combinations in each of the regions in Table 2. We think that these PAC and VAC are significant and will illustrate the value of the pathogenicity association analysis. Pathogenicity-association data showed the same general trends as did virulence frequency data.

Pathogenicity to LR1(TC) and LR2A(TC); i.e., $A_1$:$A_{2A}$ and $V_1$:$V_{2A}$ (Item 1, Table 2) was highly associated but virulence association was low in the three central regions, and the northwest region; PAC and VAC for this pair were similar and mid-range in the southeastern region. For the nine isolates in the New England sample $PAC_{1\ 2A}$ was 1.00 but $VAC_{1\ 2A}$ was 0.67. Pathogenicity to LR1(TC) and LR2D(PL) (Item 2, Table 2) showed similar trends in all regions except in the western region. Pathogenicity association and virulence association to LR1(TC) and LR10(TC) also showed regional differences. The relatively high $VAC_{1\ 10}$ for the three eastern regions was probably a result of a requirement of combined virulence to these lines to attack Blueboy (5, 19), particularly in the southeastern region. Neither pathogenicity nor virulence to LR1(TC) and LR3(TC) (Item 3, Table 2) was associated to any extent in the three central regions, but $PAC_{1\ 3}$ and $VAC_{1\ 3}$ were equal or nearly equal within each region. This indicated that very few isolates had avirulence to both LR1(TC) and LR3(TC).

Pathogenicity to LR1(TC) and LR3B(TC) (Item 6, Table 2); LR16(TC) and LR17(TC) (Item 9, Table 2); LR16(TC) and LR18(TC) (Item 10, Table 2); and LR17(TC) and LR18(TC) (Item 11, Table 2) was highly associated and virulence to the same pairs was associated infrequently. This indicates that the genes in these lines would be appropriate combinations of genes for low reaction to use in control of wheat leaf rust, at least in some regions. Both pathogenicity and virulence to LR3(TC) and LR10(TC) (Item 7, Table 2) was highly associated in all regions, except in the western region.

## DISCUSSION

Analysis of data from surveys of pathogenicity in populations of cereal rust fungi by calculating virulence frequencies (3) has become a common practice, having first been done by Samborski (21). Virulence frequencies to single lines show the potential value of certain genes for low reaction, if used singly, in control of disease. In most cereal rust systems, few single genes for low reaction will give complete protection against parasite populations. In the wheat leaf rust system in the United States, only *Lr19* will give such protection with the present parasite populations (Browder and Eversmeyer, *unpublished*).

Therefore, control programs using specific resistance must depend on deployment of cultivars with combinations of genes for low reaction. The most direct way to determine useful combinations of genes for low reaction is to analyze data from pathogenicity surveys by the pathogenicity association method presented here. As calculated in this study, PAC and VAC when taken together and interpreted, gives nearly complete information concerning the relationship of a pair of lines (or the genes they represent) and the parasite population represented by the sample used to arrive at that PAC and VAC. The difference between PAC and VAC for a given pair indicates the frequency of the population that had avirulence to both of the lines. The difference between 1.00 and the value of PAC (1.0−PAC) indicates the portion of the population which attacks one or the other of the two lines. This difference indicates only the total $A_A$:$V_B$ and $V_A$:$A_B$. If virulence frequency to A and B is known, frequencies for $A_A$:$V_B$ and $V_A$:$A_B$, if needed, also can be calculated by subtraction. Thus, PAC and VAC portray the portion of the population that will attack none, one, or two lines of a pair.

The nature of these coefficients is such that desirable gene combinations for disease control are characterized by high PAC and low VAC. In such a case, the greatest part of the population would be avirulent to both lines.

A low PAC and a low VAC for a combination would indicate that most of the protection afforded by the combination would be by only one or the other of the two genes in the combination, rather than by both genes as when PAC is high and VAC is low. A combination with a high PAC and a high VAC would be of very little value in control of disease, because most of the population would attack such a combination and only a small portion or none would be avirulent to both lines in the combination. The method of calculating these coefficients precludes a high VAC and a low PAC.

It is axiomatic, within the gene-for-gene relationship, that if a cultivar has two genes for low reaction, then a parasite must have the two corresponding genes for high pathogenicity to attack it. Blueboy has been shown to have *Lr1* and *Lr10* (5). The necessity for *P. recondita* genes for high pathogenicity at *PLr1* and *PLr10* to attack Blueboy is reflected in the $VAC_{1\ 10}$ of 0.62 in the southeastern region (Item 4, Table 2) and virulence frequencies of 83% and 73% to LR1(TC) and LR10(TC),

respectively (Table 1), and their relationship to Blueboy. According to current information, a similar conclusion cannot be drawn about virulence to LR3(TC) and the cultivars grown in many regions of the United States (Table 1). The allele for high pathogenicity at *PLr3* appears to be a relict and unnecessary gene in these regions. However, our knowledge of the reaction genotype to *P. recondita* of commercially grown cultivars is sparse. High virulence frequencies or a high VAC to lines with specific genes is an indication at least that those host genes for low reaction may be present in commercially grown cultivars.

The analyses presented in this paper deal with combinations of pathogenicity to sets of lines taken one or two at a time. The same principles may be applied to analysis of pathogenicity to sets of lines taken more than two at a time. Increase of numbers within combinations would be useful in determining effective combinations of genes for controlling leaf rust of wheat. Numbers of lines within combinations should be limited to the number of genes which can be feasibly manipulated in a breeding program.

The pathogenicity association analysis presented here is a marked departure from classic pathogenic race analysis. Pathogenic race analysis accounts pathogenicity only for combinations of $n$ lines taken $n$ at a time (number of possible combinations is one) and this is its weakness. With this method, the relationship of one line to another cannot be studied easily. Our method accounts pathogenicity to $n$ lines taken $r$ at a time; number of possible combinations:

$$C_{(n,r)} = \frac{n!}{r!\,(n\text{-}r!)}$$

This method shows much more detailed information for guiding breeding programs, but this information is obviously more difficult to transmit. The analysis is systematic, but has no nomenclatural facility. Genes rather than races are emphasized; genes are the biologically functional units (3). The focal point of the parasite becomes fungal cultures with *known differential pathogenicity* that can be used in manipulating host genes for low reaction in breeding programs. Two cultures, one with $A_AV_B$ and the other with $V_AA_B$, are needed to easily combine the two genes A and B. Cultures are controllable, experimental entities rather than abstract taxonomic groups represented by race names. With these concepts, the pathogenicity formula in the system proposed by Loegering and Browder (17) becomes a description for cultures rather than a nomenclatural system for races.

Wolfe et al. (24) have independently arrived at a similar method of pathogenicity survey data analysis. They propose that combinations of host genes for low reaction to which there is significantly lower observed than expected frequency of combined virulence are the appropriate combinations to be used in control. The expected frequency of combined virulence is the product of the observed virulence frequency to each line in the combination. Our method provides a way of portraying combined virulence to a pair, the VAC, but also places emphasis on combined avirulence frequency (PAC - VAC).

We think that the pathogenicity association analysis represents a simplification of analyzing data from pathogenicity surveys although it appears more complex because of the volume of the reduced data and the lack of a race nomenclatural system.

## LITERATURE CITED


1. ANONYMOUS, 1959. A proposed modification of the system of wheat leaf rust race identification and nomenclature. (A Committee of North American Wheat Leaf Rust Research Workers). Plant Dis. Rep. 43:613-615.
2. ANONYMOUS, 1961. The North American 1961 set of supplemental differential wheat varieties for leaf rust race identification. (A Committee of North American Wheat Leaf Rust Research Workers). Plant Dis. Rep. 45:444-446.
3. BROWDER, L. E. 1971. Pathogenic specialization in the cereal rust fungi, especially Puccinia recondita f. sp. tritici: concepts, methods of study, and application. U.S. Dep. Agric. Tech. Bull. 1432. 51 p.
4. BROWDER, L. E. 1973. Specificity of the Puccinia recondita f. sp. tritici:Triticum aestivum 'Bulgaria 88' relationship. Phytopathology 63:524-528.
5. BROWDER, L. E. 1973. Probable genotype of some Triticum aestivum 'Agent' derivatives for reaction to Puccinia recondita f. sp. tritici. Crop Sci. 13:203-206.
6. BROWDER, L. E., and M. G. EVERSMEYER. 1976. Recording and machine processing of cereal rust infection-type data. Plant Dis. Rep. 60:143-147.
7. BROWDER, L. E., and H. C. YOUNG, JR. 1975. Further development of an infection-type coding system for the cereal rusts. Plant Dis. Rep. 59:964-965.
8. DAY, P. 1974. Genetics of host-parasite interaction. W. H. Freeman and Co., San Francisco. 238 p.
9. DYCK, P. L., and E. R. KERBER. 1971. Chromosome location of three genes for leaf rust resistance in common wheat. Can. J. Genet. Cytol. 13:480-483.
10. DYCK, P. L., and D. J. SAMBORSKI. 1968. Genetics of resistance to leaf rust in the common wheat varieties Webster, Loros, Brevit, Carina, Malakof, and Centenario. Can. J. Genet. Cytol. 10:7-17.
11. DYCK. P. L., and D. J. SAMBORSKI. 1968. Host-parasite interaction involving two genes for leaf rust resistance in wheat. Pages 245-250 *in* Proc. Third Int. Wheat Genet. Symp., 5-9 August, 1968, Canberra, Australia. 479 p.
12. GREEN, G. 1965. Stem rust of wheat, rye and barley in Canada in 1964. Can. Plant Dis. Surv. 45:23-29.
13. JOHNSON, R., R. W. STUBBS, E. FUCHS, and N. H. CHAMBERLAIN. 1972. Nomenclature for physiologic races of Puccinia striiformis infecting wheat. Trans. Br. Mycol. Soc. 58:475-480.
14. JOHNSTON, C. O., R. M. CALDWELL, L. E. COMPTON, and L. E. BROWDER. 1968. Physiologic races of Puccinia recondita f. sp. tritici in the United States from 1926 through 1960. U.S. Dep. Agric. Tech. Bull. 1393. 18 p.
15. JOHNSTON, C. O., and E. B. MAINS. 1932. Studies on physiologic specialization in Puccinia triticina. U.S. Dep. Agric. Tech. Bull. 313. 22 p.
16. KERNKAMP, M. F. 1965. Pathogenic specialization in relation to taxonomy. Phytopathology 55:821-822.
17. LOEGERING, W. Q., and L. E. BROWDER. 1971. A system of nomenclature for physiologic races of Puccinia recondita tritici. Plant Dis. Rep. 55:718-722.
18. LOEGERING, W. Q., and C. H. BURTON. 1974. Computer-generated hypothetical genotypes for reaction and pathogenicity of wheat cultivars and cultures of

Puccinia graminis tritici. Phytopathology 64:1380-1384.
19. NELSON, L. R., D. D. MOREY, and A. R. BROWN. 1972. Severe leaf rust on wheat in Georgia. Plant Dis. Rep. 56:534-535.
20. ROELFS, A. P., and D. M. MC VEY. 1972. Wheat stem rust races in the Yaqui valley of Mexico during 1972. Plant Dis. Rep. 56:1038-1039.
21. SAMBORSKI, D. J. 1966. Leaf rust of wheat in Canada in 1965. Can. Plant Dis. Surv. 46:33-35.
22. SOKAL, R. R., and P. H. A. SNEATH. 1963. Principles of numerical taxonomy. W. H. Freeman and Co. San Francisco and London. 359 p.
23. WATSON, I. A., and N. H. LUIG. 1961. Leaf rust on wheat in Australia: a systematic scheme for the classification of strains. Proc. Linn. Soc. N.S.W. 86:241-250.
24. WOLFE, M. S., J. A. BARRETT, R. C. SHATTOCK, D. S. SHAW, and R. WHITBREAD. 1976. Phenotype-phenotype analysis: field application of the gene-for-gene hypothesis in host-pathogen relations. Ann. Appl. Biol. 82:369-374.
25. YOUNG, H. C., JR., and L. E. BROWDER. 1965. The North American 1965 set of supplemental differential wheat varieties for identification of races of Puccinia recondita tritici. Plant Dis. Rep. 49:308-311.