GENETICS OF PATHOGENICITY OF *PUCCINIA GRAMINIS TRITICI* ERIKS. & E. HENN.

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CHAPTER I

INTRODUCTION

Puccinia graminis Pers. F. sp. tritici Eriks. & Henn. is the causal agent of the stem rust disease of wheat Triticum L. sp. barley, Hordeum vulgare L. and other grasses. (8) The stem rust derives from the common occurance of the disease on the stem of the wheat plant, although it also occurs on awns, glumes, leaves, and sheaths. The disease appears as red pustules urediospore stage and as black pustules in the teliospore stage. (17) The sexual stage of the fungus occurs on the common barberry Berberis vulgaris L.

At one time or another stem rust epidemics have occured almost every place wheat is grown. Tremendous losses in wheat production have resulted. After the epiphytotic in the northern plains in 1904, one of the first departments of plant pathology was founded in the United States at the University of Minnesota. Another major epiphytotic in 1916 was estimated to have reduced the yield potential for that year by 38%.

The federal and state governments took steps to combat this disease with many programs of research and breeding as well as the initiation of the barberry irradication campaign. (17) Even before the development of more scientific agriculture German farmers knew that there was a relationship between the presence of barberry and the rusted condition of their wheat fields. (5)

It has been shown that the stage of host maturity when the rusting occours, greatly affects the amount of damage to crop yield. For instance, it is estimated that an infection of 10% when the wheat is in the boot stage will result in a 100% loss by harvest time, but if the same wheat has a 10% infection in the hard dough stage it will only have a loss of about 5% in yield. For this reason the early inoculation of wheat from barberry was very important. (1) Also since the sexual stage of Puccinia graminis occours on barberry it is very important in providing genetic variation in this pathogen. (5)

Early work with rusts (8) demonstrated that pathogenicity in stem rust was controlled by independent genes. Later Flor (3) demonstrated that in the flax and flax rust relationship there is a one to one correlation between the host's genes for reaction and the pathogen's genes for pathogenicity. Loegering (11) coined the term aegricorpus to describe the infection site; a production of the genetics of both pathogen and host. Neither the host nor the parasite can determine the appearance of the aegricorpus alone, yet it reveals the genetics of both host and parasite.

The purpose of this study was to determine the genes for pathogenicity in an F_2 population of P. graminis f. sp. tritici using the gene for gene concept of plant disease genetics. F_2 cultures from a cross of culture 111-55A with culture 36-55A were used to inoculate 15 lines of wheat to determine the apparent genetic ratios of low vs. high pathogenicity. (10)

CHAPTER II

LITERATURE REVIEW

The first major studies concerning the genetics of pathogenicity began with Flor's studies of flax rust or the Linum sp. - Melamspora lini association. From those studies the current gene for gene concept emerged as a model for host-parasite interactions in plant disease. (3) Similar studies with other organisms support this concept, and it is possible that it is a universal pattern. (4) (15) (16)

P. graminis tritici has been a focal point of genetic studies, using its pathogenicity on Triticum species as a genetic character. The gene for gene pattern appears to link this parasite and host in a form of symbiosis. (11)

Essentially, the gene for gene concept of plant disease genetics is of a host-parasite system in which genes for pathogenicity in the parasite have corresponding genes in the host which determine host reaction. The corresponding genes in the two organisms interact to produce an infection type. These two corresponding gene pairs interact to give the infection type, so long as other factors or other corresponding gene pairs do not intervene. In actual host-parasite interactions two or more corresponding gene pairs are usually acting at the same time. In most cases the corresponding gene pair with the lowest infection type is the highest infection type that can be expressed. (11)

Loegering and Powers (11) presented a genetic model for the gene for gene system; an extension of Persons mathematical model. They explained diagramatically what they called the four categories of interaction. The first two categories I and II are interactions between alleles at a single locus and between gene pairs at different loci, respectively, within a single organism. Categories III and IV are of the interorganismal type. Category III interactions are those between corresponding genes for pathogenicity and reaction in the parasite and host respectively. Category IV interactions are interactions between sets of corresponding gene pairs. (11) In this situation , only those genes for pathogenicity in the parasite that have corresponding genes for low reaction in the host can be detected, and conversly, only those genes for reaction in the host that correspond to genes for low pathogenicity in the parasite can be detected. Otherwise the infection type is always high. Using this system one can get a measure of genetic ratios by using an F_2 segregating population of either the host or the parasite and a homogeneous population of the other (preferably monogene lines) to determine infection types and therefore the genetics involved. Using the quadratic check of Rowel et al (20) greatly simplifies the determination of the number of genes involved.

Hybridization is an important factor in the development of race populations in those pathogens having a sexual cycle. In Flor's studies of a cross of races six and 22 of M. lini 54 pathogenically different cultures were identified in a population of 67 F_2 cultures. The two parents varied pathogenically on 12 different monogenic host lines. Theoretically, if enough F_2 cultures of this cross had been studied 2^{12} or 4,096, different cultures could have been identified. The origin of new races or combinations of pathogenicity genes by hybridization is

limited however to the reassortment of existing genes for pathogenicity.

Mutation, on the other hand, provides new gene types, and therefore produces races with pathogenicity beyond the existing gene pool. Mutations at given loci generally are rare, but considering the numbers of spores produced by pathogens such as those causing rust diseases, perhaps such mutations occur often enough to be significant in pathogen gene pool development. Pathogenicity has generally been found to be recessive, so that if dominance is complete a two point mutation will be necessary before the gene can be expressed, or an F_2 generation derived from hybridization would be required. This has been shown by Rowell et. al. (20) who demonstrated that a largely heterozygous individual has a far greater chance of producing a new pathogenicity character.

Parasexualism, another method of producing variation with out the traditional sexual processes has been reported to result from hyphal fusions and exchange of nuclei in P. graminis tritici when mixtures of pathogenically different urediospores were used for inoculation. (2) (23)

In the field pathogen populations seem to be controlled by two major factors: 1.) specific genes for pathogenicity relative to host populations, and 2.) relative agressiveness and fitness or survival ability. (18) Those two factors may involve the same or linked loci, and there is evidence that heterosis may be involved in agressiveness of a pathogen in much the manner as it effects higher plants. (25)

CHAPTER III

MATERIALS AND METHODS

In this study 114 cultures of P. graminis tritici were used to inoculate 15 different lines of wheat. The cultures of P. graminis tritici used were the parents of a cross 111-55A, and 36-55A, a culture of the F. of this cross, and 111 cultures of the F_2 progeny resulting from selfing the F. (11) (19) (21) Some of the original cultures were lost and new ones were collected, but were suspected of being mutants from the original cultures. (6) Loegering and Power's (11) original study of these cultures showed at least eight genes for low pathogenicity involved, all of which segregated in a Mendelian manner. Williams, et. al. (24) demonstrated that the gene for gene system was expressed when these same cultures and wheat derived lines were used.

The 15 lines of wheat used included three "universal susepts" (16) Chinese, Little Club, and W2691. These three lines were used as controls for the other 12 wheat lines; being a reference for the highest infection type in the experiment.

Those lines for which Chinese was used as a control were:
ISr 7b-Ra (C. I. - 14165) - Selection from a cross of Hope X Chinese with five backcrosses to Chinese monosomic for chromosome 4B. backcrossed to Chinese two times and selfed ten times.
ISr 8-Ra (C. I. - 14167) - Selection from a cross of Red Egyptian X Chinese with four backcrosses to Chinese monosomic for chromosome 6A,

backcrossed to Chinese two times and selfed ten times.

ISr 9a-Sa (C. I. 14170) - Selection from a cross of Red Egyptian X Chinese with three backcrosses to Chinese monosomic for chromosome 2B, backcrossed to Chinese two times and selfed 11 times.

ISr 18-Ra (C. I. - 14179) - Selection from a cross of Hope X Chinese backcrossed to Chinese monosomic for chromosome ten, backcrossed to Chinese two times and selfed 12 times. (9)

Those lines for which Little Club was used as a control were:

SrU.-F_3 selection from a cross of Red Egyptian 2D X Little Club.

Marquis A, B, and C - three lines, each monogenic for different genes for low reaction from the line Marquis.

Reliance A, and C - two lines, each monogenic for different genes for low reaction from the line Reliance.

These Marquis and Reliance derived lines were developed by Williams, Gough, and Rondon, (24) by crossing Marquis and Reliance with Little Club. The F_1's were backcrossed to Little Club and then selections were made in the F_2 and F_3 for monogenic reaction to P. graminis tritici culture 111-552. Each parental line segregated for three low reaction genes.

Those lines for which W2691 was used as controls were: Kota 0; and 2. These two lines were selected from a cross of Kota X W2691. Both genes for low reaction are located on chromosome 2BL. The gene in Kota 0 is possibly the same as the gene Sr 28. Kota 2 may be allelic to Sr 16. (14)

All of the pathogen cultures and host lines were supplied by Dr. W. Q. Loegering, Department of Plant Pathology, University of Missouri, Columbia.

Twenty seeds of each of these 15 lines of wheat were planted in 5 cm pots. One pot of each wheat line was planted for each culture of P. graminis

tritici used. Six sets of pots were planted each day. The seedlings were allowed to grow on the green house bench for seven to eight days, or until the second leaf appeared. They were prepared for inoculation by gently rubbing the primary leaves wet, between the thumb and forefinger to break the surface tension of the leaves. The leaves were then atomized with distilled water. While still wet the plants were dusted with a mixture of two mg of spores and 25 mg of talcum. The inoculated plants were placed in moist chambers and allowed to stand over night. (22) The next morning the moist chamber lids were opened allowing the plants to dry off slowly before removing them from the chamber. The plants were then placed on the green house bench in full light at 22^0 C $\pm$ 5. Thirteen to fourteen days after inoculation most of the plants were ready for observation, and notes were recorded on infection type. The H-L system of evaluation of infection types devised by Loegering, McIntosh, and Burton (10) was used to catagorize pathogenicity of the cultures. Also, an individual infection type was recorded according to the scale of Stakeman et. al. (21), if it was different from the control.

CHAPTER IV

RESULTS

In gene for gene systems in general and in the P. graminis tritici--Triticum sp. system in particular low reaction and low pathogenicity are genetically dominent over high. However, the F_2 cultures of P. graminis tritici from the cross of culture 111-55A X culture 36-55A segregated in a manner that would not be expected with this hypothesis on several of the lines of wheat used. On eight of the supposed monogenic wheat lines the F_2 cultures segregated with a single gene ratio as would be expected, but only on five did the cultures segregate with the typical three lows: one high. (Table I)

Those wheat lines on which the F_2 cultures segregated with a good fit to a 3:1 ratio were: ISr8-Ra, 79 low(L) to 32 high (H) ($.30<P<.50$). ISr8-Ra was the only line with Chinese as a control in which the segregation of the cultures fit a 3:1 ratio. The cultures segregated with a good fit to 3:1 ($.50<P<75$) on: Kota 0;, 81 (L) to 30 (H); on SrU ($.95<P<97$), 83 (L) to 28 (H), on Marquis A ($.75<P<.90$); and on Marquis C ($.50<P<.75$), 81 (L) to 30 (H). Eighty nine per cent of the F_2 cultures had the same designation for Marquis A and Marquis C using the low high system, although there was often considerable difference in the actual low infection type. For example F_2 culture number 143 with Marquis A gave a "0;" infection type but with Marquis C produced a "2=". This variation was clearly discernible.

Those lines on which the F_2 cultures segregated with a low: three high ratio (the reverse of the typical response) were: ISr76-Ra, on which the cultures segregated 20 (L) to 91 (H) ($.05<P<.10$); Marquis B on which the cultures segregated 25 (L) to 86 (H) ($.5<P<.75$); Reliance A on which the cultures segregated 25 (L) to 75 (H) (.99). There were only 100 F_2 cultures recorded for Reliance A because of the relatively high mortality rate of the Reliance A seedlings.

The F_2 cultures showed apparent two-gene segregations on the remaining four wheat lines. ISr7b-Ra was listed with those lines on which the F_2 cultures segregated one low:three high, but the fit was rather poor. Pathogenicity on this line probably should be considered to involve two genes since the segregation of 20 (L) to 91 (H) actually has a higher P value for a 3:13 ratio ($.30<P<.50$) than for a 1:3 ratio. The segregation on two of the remaining wheat lines fit a two gene 15:1 ratio. On ISr9a-Sa the cultures segregated were 100 (L) to 11(H) ($.10<P<.20$) and on Reliance C 100 (L) to 4 (H) also a good fit to a 15:1 ratio ($.25<P<30$). The cultures segregated on ISr18-Ra 66 (L) to 45 (H) a good fit to a 9:7 ratio ($.50<P<.75$). On Kota "2" the cultures segregated with a good fit to a 7:9 ratio 50 (L) to 61 (H) ($.75<P<.95$).

On the control cultivar, Chinese, the F_2 cultures segregated 77 (L) to 34 (H), a good fit to a 3:1 ratio ($.10<P<.20$). This segregation undoubtedly had an effect on those ratios which were compared to it as a High infection type standard. In some cases the infection type on Chinese was as low as a "0;". The other two controls did not evidence any segregation among the F_2 cultures, however environmental variability did cause fluctuation in the infection types recorded on all wheat lines, probably because the entire experiment was done in the greenhouse where temperature control was not precise.

Other than with Marquis A and Marquis C on which 99 of 111 F_2 cultures had the same infection type using the Low High system, there was no significant correlation between cultures on the other wheat lines.

TABLE I

SEGREGATION OF F_2 CULTURES FROM THE CROSS OF CULTURE 111-55A X CULTURE 36-55A OF *PUCCINIA GRAMINIS* F. SP. *TRITICI* ON 15 CULTIVARS AND LINES OF WHEAT *TRITICUM SP.*

Wheat Line	No. of F_2 Culture Segregates[a] Observed Low	Observed High	Expected Low	Expected High	Proposed Ratio	X^2 Value	P. Value between
Chinese[b]	77	34	83.25	27.75	3:1	1.8768	.10 - .20
ISr76-Ra	20	91	27.75	83.25	1:3	2.8858	.05 - .10
ISr76-Ra[c]	20	91	20.8125	90.1875	3:13	.039039	.75 - .90
ISr8-Ra	79	32	83.25	27.75	3:1	.86786	.30 - .50
ISr9a-Sa	100	11	104.0625	6.9375	15:1	2.53753	.10 - .20
ISr18-Ra	66	45	62.4375	48.5625	9:7	.464607	.50 - .75
W2691b	No segregation						
Kota "0;"	81	30	83.25	27.75	3:1	.1418918	.50 - .75
Kota "2"	50	61	48.5625	62.4375	7:9	.0756469	.75 - .90
Little Club[b]	No segregation						
Marquis A	84	27	83.25	27.75	3:1	.0270269	.75 - .90
Marquis B	25	86	27.75	83.25	1:3	.3633633	.50 - .75
Marquis C	81	30	83.25	27.75	3:1	.2432432	.50 - .75
Reliance A	25	75	25	75	1:3	0.	.99 -1.00
Reliance C	106	4	103.125	6.875	15:1	1.282424	.25 - .30
SrU	83	28	83.25	27.75	3:1	.0030029	.95 - .97

a Segregates classified for Low (L) and High (H) pathogenicity

b Control for the wheat lines listed beneath

c ISr76-Ra has two proposed ratios, the second having the highest probability.

CHAPTER V

DISCUSSION

Infection levels attained with most of the F_2 cultures were satisfactory. Although there were a few plants escaped infection there were no complete failures. Some of the cultures showed a lack of agressiveness in terms of the number of pustules formed and the length of time required for pustules to mature. This apparent lack of agressiveness may be due to the same phenomenon discussed by Martin and Ellingboe (13) involving the interaction of a pathogen's genes for "high" pathogenicity and the host's genes for "low" reaction.

Segregation of the F_2 cultures on Chinese was expected, because Chinese has a low reaction gene, Sr 9f, which corresponds with a low pathogenicity gene segrating in the F_2 cultures of P. graminis tritici used. The interaction of these corresponding genes is temperature sensitive. (7) The effect of Sr 9f was evident in the wheat lines that had Chinese as a control and may test the validity of the proposed ratios, although it must be noted that fits were good to their ratios proposed. ISr8-Ra was the only line derived from crosses with Chinese cultures segregated with the expected 3:1 ratio. The cultures segregated in two gene ratios in all other lines obtained from crosses with Chinese. On ISr7b-Ra, the ratio was a satisfactory fit to a 3:13 ratio which indicated that one of two genes for "high" pathogenicity was incompletely dominant. Segregation on ISr18-Ra gave an unexpected ratio of about 9:7, which indicates an

accumulative effect of two genes, or may be the result of a distorted ratio. (12) On the remaining line ISr9-Sa the cultures segregated in a 15:1 ratio.

The cultures did not segregate on the control cultivar W2691, but on Kota 0; the cultures segregated in the expected 3:1 ratio. On Kota "2" however the F_2 cultures segregated with a good fit to a 7:9 ratio. This did not agree with Rondon. (19) The Kota "2" line was supposed to be the same so far as low reaction genes were concerned as his K±C which had only one gene for low reaction and produced a 3:1 ratio using the same F_2 cultures. This would imply a second gene in Kota "2" in addition to one in common with K±C.

The F_2 cultures did not segregate on Little Club, the control for the Marquis and Reliance lines and SrU. Williams, et. al. (24) developed the Marquis and Reliance lines from crosses with Little Club and tested them using these same F_2 cultures. Their results verified the gene for gene relationship, suggested by Loegering and Powers (11) who developed the F_2 cultures and tested them on Marquis and Reliance, There was considerable difference in the results obtained with the Marquis and Reliance lines in this study and those reported by Williams, et. al. (24). The cultures segregated in a single gene ratio of 3:1 on Marquis A, but in a 1:3 on Reliance A. Williams et. al. (24) reported a 3:1 ratio on Reliance A, which later shown to have the same gene as Marquis A and no segregation at all on Marquis B, which in this study the cultures segregated 1:3. On Marquis C the cultures segregated 3:1 and on Reliance C segregated 15:1. Again these results did not agree with Williams et. al. (24). They got identical segregations of 3:1 on both Marquis C and Reliance C indicating the same gene was present. In this experiment 99 of 111 F_2 cultures gave the same infection type on Marquis A and Marquis C indicating they may

carry the same gene, which again did not agree with Williams et. al. (24). These F_2 cultures segregated on SrU. with a very good fit to a 3:1 ratio. Some of the cultures on SrU produced some infection types which were higher than those on Chinese. This line therefore could not have the Chinese gene Sr9F, for low reaction which is probably in Red Egyptian one of SrU's parental lines.

It was unusual that there were five out of 13 lines on which the segregation indicated that high pathogenicity was dominant, or at least partly so. Most genetic studies with this and other pathogens have indicated that low pathogenicity is almost always dominant. This would warrant some further investigation, as would the discrepencies between the results reached here and those of Williams, Gough, and Rondon. (24)

CHAPTER VI

SUMMARY

1. Eleven cultures of an F_2 population of Puccinia graminis tritici were used to inoculate 15 lines of wheat in a test for segregation with respect to genes for pathogenicity. The infection types were rated using the high-low system with a set of controls.
2. The F_2 cultures segregated with both one and two gene ratios on the wheat lines tested. Of the 13 wheat lines used, eight produced single gene ratios and five produced two-gene ratios with these F_2 cultures.
3. Most of the segregations showed low pathogenicity to be dominant, at least partially. Again, eight lines produced ratios indicating that low pathogenicity dominant, while in the other five lines high pathogenicity was dominant.
4. Ratios of 3:13, 7:9, and 9:7 were obtained with these cultures on certain wheat lines. Such ratios may indicate category II or possibly category IV interactions were involved.

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