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HOST-PARASITE INTERACTION IN FLAX RUST—ITS GENETICS AND OTHER IMPLICATIONS [1]

H. H. Flor [2]

SUMMARY

The pathogenicity of 67 F_2 cultures of a cross of race 6 with race 22 of the flax rust pathogen was determined on 32 varieties of flax that had been selected as carrying single genes for rust reaction. Two varieties were resistant and 6 were susceptible to all F_2 cultures. On 23 of the 24 varieties on which the F_2 cultures segregated for pathogenicity, the ratio of avirulent to virulent cultures approximated the 3:1 expected if virulence on each was conditioned by a pair of recessive genes. On Ottawa 770B, 2 pairs of genes may have conditioned pathogenicity. Fifty-four pathogenic races were identified from the 67 cultures.

Host–parasite interaction in flax rust may be explained by assuming a gene-for-gene relationship between rust reaction in the host and pathogenicity in the parasite. Pustule type, the criterion of both reaction and pathogenicity, is conditioned by specific pairs of genes, one in the host and the other in the parasite. In flax and the flax rust fungus, 25 such pairs of genes have been identified.

Because of the gene-for-gene relationship between reaction in the host and pathogenicity in the parasite, the recessive gene complement of a uredial clone (culture) is established by determining its pathogenicity on differential varieties with single rust-conditioning genes. The homozygosity or heterozygosity of the dominant genes is established by selfing the uredial clone. Thus, a method for identifying the pathogenic genotype of races of the rust fungi has been devised. This makes possible the use of the biotype as the basic concept of race.

The gene-for-gene relationship of rust reaction and pathogenicity in host and parasite facilitates the development of rust-resistant varieties and opens up new approaches to studies of the origin of new races, mutation for rust reaction in the host and pathogenicity in the parasite, the evaluation of epidemiology data, and the nature of resistance.

Notwithstanding extensive investigations on physiologic specialization, pathogenic variability, the nature of resistance, and epidemiology of the cereal rusts, newly developed rust-resistant varieties of wheat, oats, and flax often succumb to rust after a few years of commercial production. This suggests the need for a new approach to studies on the rusts.

In 1942, Flor (4) pointed out the gene-for-gene relationship between pathogenicity in the flax rust fungus, *Melampsora lini* (Pers.) Lév. and rust reaction in the host, *Linum usitatissimum* L. Later studies not only verified this finding, but also showed the relationship to be highly specific.

F_2 cultures of race 6 × race 24 segregated for pathogenicity on the varieties Buda, Akmolinsk, and Bombay (3). Virulence was recessive. On Akmolinsk and Bombay, varieties having 1 gene for rust reaction, pathogenicity was conditioned by single genes. On Buda, which has 2 genes for rust reaction, pathogenicity was conditioned by 2 genes. The gene for pathogenicity on Akmolinsk appeared to be linked with 1 of the genes for pathogenicity on Buda.

The parent races in the cross of race 22 with race 24 attacked 15 of 16 differential varieties (4). F_1 cultures attacked only the 3 differentials susceptible to both parent races. From 133 F_2 cultures, 64 pathogenically different races were identified. Virulence was recessive and conditioned by 1 gene on Abyssinian, Akmolinsk, Bombay, Kenya, Leona, Newland, Ottawa 770B, Pale Blue Crimped, and Tammes Pale Blue, varieties having 1 gene for resistance to the avirulent parent race. Two genes conditioned virulence on Bolley Golden and Italia Roma, varieties having 2 genes for resistance to race 24, and 3 genes conditioned virulence on Morye, a variety having 3 genes

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for resistance to race 24. The genes for virulence on Abyssinian, Akmolinsk, and Leona, and on Kenya and Pale Blue Crimped were so closely linked as to be inherited as units. These studies indicated that 1 of the genes for virulence on Bolley Golden was identical or closely linked with 1 of the genes for virulence on Italia Roma. Likewise, the genes for virulence on Tammes Pale Blue either were identical or closely linked with 1 of the 3 pairs of genes conditioning virulence on Morye.

Although the pathogenicity of 98 F_2 cultures secured from 3 populations of the cross of race 6 with race 22 was determined (4), that number of cultures was considered too small for statistical analysis of the interaction of the genes for pathogenicity. Differences in the segregating ratios between the race 22 × race 24 cultures and the race 6 × race 22 cultures on Tammes Pale Blue and Morye, however, emphasized the gene-for-gene relationship between reaction in the host and pathogenicity in the parasite. In the cross of race 22 × race 24, pathogenicity on Tammes Pale Blue and Morye was conditioned by 1 and 3 genes, respectively. In the cross of race 6 × race 22, pathogenicity on Tammes Pale Blue was conditioned by 2 genes and pathogenicity on Morye by 4 genes. The Koto gene had not been discovered, but subsequently (6) it was determined that both Tammes Pale Blue and Morye carry the Koto gene. Koto is susceptible to races 22 and 24 and resistant to race 6. Consequently, the Koto gene in Tammes Pale Blue and Morye was not effective against the race 22 × race 24 hybrids but was effective against the race 6 × race 22 hybrids.

MATERIALS AND METHODS.—Studies on the genetics of pathogenicity in the rusts are laborious and exacting. Statistically significant data on the interaction of genes for virulence can be secured from fewer cultures if the differential varieties possess single rust-conditioning genes than if they possess 2, 3, 4, or more. Consequently, a new series of flax rust differentials was developed (6). Each new differential apparently possesses a single gene for reaction to North American races. The new differentials were selected on the basis of the possesssion of genes that 1) condition resistance to North American races, 2) condition reaction in commercial varieties, and 3) identify races on a world-wide basis.

Telia developed from the F_1 culture of race 6 × race 22, Hybrid A, were found to be viable after 7 years of storage at about 3°C, and 67 F_2 cultures were secured. In pathogenicity studies on the rusts, the interaction is between the dikaryotic (2n) urediospore culture and the diploid (2n) host. Because the urediospore is the repeating and pathogenic phase of the flax rust fungus, it has an advantage in genetic studies over the smuts and higher plants in that each F_2 variant may be maintained indefinitely as a uredial clone by periodic passage through the host. As the new lines of flax possessing single rust-conditioning genes were developed, their reaction to the 67 F_2 cultures was determined. These results were verified in a final test of the reaction of 32 varieties to each culture.[3] Although some of these varieties do not serve to identify numbered races, all that differentiate the F_2 cultures are of equal importance in a study on the genetics of pathogenicity.

RESULTS.—*Pathogenicity studies.*—The reaction of 32 varieties of flax to 67 F_2 cultures was determined. Two varieties, Bombay and J.W.S., were resistant to all cultures; and 6 varieties, Akmolinsk, Barnes, Bison, Victory A, Wilden, and Williston Brown, were susceptible to all cultures. The 24 varieties on which the cultures segregated for pathogenicity fell into 12 differential groups (Table 1). Based on a dichotomous classification, 54 pathogenic races were identified among the 67 F_2 cultures. Theoretically, 2^{12} or 4096 races were obtainable from this cross if enough cultures had been studied.

On all the differential varieties except Ottawa 770B, the cultures segregated into ratios satisfactorily approximating the 3 avirulent to 1 virulent expected if virulence on each was conditioned by a single pair of recessive genes. This is further confirmation of the possession by each of these varieties of a single gene for resistance to North American race 6.

The occurrence of 7 virulent to 60 avirulent cultures on Ottawa 770B is difficult to explain on a simple Mendelian basis. In tests with the cross of race 22 × race 24, 33 of 133 F_2 hybrid cultures attacked Ottawa 770B (4), almost a perfect 1:3 ratio. In the previous tests of race 6 × race 22 (4), 10 of 74 F_2 cultures of Hybrid A, 1 of 16 F_2 cultures of Hybrid B, and 2 of 8 F_2 cultures of Hybrid C attacked Ottawa 770B. Henry (7), Myers (10), and Flor (5) found that in Ottawa 770B, 1 gene conditioned resistance to North American races. Kerr (9) found, however, that in addition to the *L* gene which conditions resistance to North American races, Ottawa 770B possesses a gene in the *N* series of allelomorphs conditioning resistance to some Australian races. Although the gene for avirulence on varieties possessing the *N* gene of Ottawa 770B is not known to occur in North American races, it is possible that race 6 was heterozygous for it.

The χ^2 values were calculated for association of pathogenic characters on the 12 varietal groups. These data indicated that pathogenicity on 9 varieties or groups of varieties was inherited independently or was too loosely linked to be evident in the analysis of the limited number of cultures studied. Pathogenicity was inherited independently on Ottawa 770B, Dakota, Birio, and Burke and was inherited as an independent unit on Abyssinian, Koto, Leona, Ward, and Wells; on Bowman, Clay, Grant, and Minnesota selection; and on Argentine selection, Cortland, and Lino 6899 M.A. Pathogenicity on Polk and Marshall was closely linked, and pathogenicity on these varieties was loosely linked with that on Cass. Pathogenicity was inherited as a unit on B. Golden selection, Kenya,

[3] Six cultures were lost before Towner (C. I. 1561) was isolated.

TABLE 1.—*Reaction of varieties and lines of flax to 67 cultures of a cross of race 6 with race 22 of Melampsora lini*

No. cultures with indicated pathogenicity	Reaction[a] on indicated differential-variety group[b]											
	1	2	3	4	5	6	7	8	9	10	11	12
1	S	S	R	R	S	R	R	R	S	S	R	S
1	S	R	S	R	S	R	R	R	R	S	R	S
1	S	R	R	S	R	R	R	R	S	S	R	S
1	S	R	R	R	S	S	S	R	S	S	R	R
1	S	R	R	R	S	R	R	R	R	R	R	R
1	S	R	R	R	R	R	R	R	S	S	R	R
1	S	R	R	R	R	R	R	R	R	R	R	R
3	R	S	S	R	R	S	S	R	R	R	R	R
1	R	S	S	R	R	R	R	S	S	S	R	R
1	R	S	S	R	R	R	R	R	R	R	S	R
1	R	S	R	S	R	R	R	S	R	R	R	S
1	R	S	R	R	S	R	R	S	S	R	R	R
1	R	S	R	R	S	R	R	R	R	R	R	R
1	R	S	R	R	R	S	S	R	S		S	R
1	R	S	R	R	R	S	S	R	R	R	R	R
1	R	S	R	R	R	R	S	S	R	S	R	R
1	R	S	R	R	R	R	S	S	R	S	R	R
1	R	S	R	R	R	R	R	R	S	S	R	R
1	R	S	R	R	R	R	R	R	R	R	R	R
1	R	R	S	S	S	R	R	S	S	R	S	R
1	R	R	S	S	R	S	S	R	R	S	S	S
1	R	R	S	S	R	S	S	R	R	R	R	S
1	R	R	S	S	R	R	R	R	S	S	R	R
1	R	R	S	R	S	S	S	S	R	R	R	R
2	R	R	S	R	S	S	S	R	R	R	R	R
1	R	R	S	R	S	R	R	R	R		R	R
1	R	R	S	R	R	S	S	R	S	R	R	S
1	R	R	S	R	R	S	S	R	R	R	R	S
1	R	R	S	R	R	R	R	S	S	S	S	R
1	R	R	S	R	R	R	R	R	S	S	S	S
1	R	R	S	R	R	R	R	R	R	R	S	S
1	R	R	S	R	R	R	R	R	R		S	R
1	R	R	S	R	R	R	R	R	R	R	R	S
1	R	R	R	S	S	S	S	R	S	S	R	R
1	R	R	R	S	S	R	R	R	R	R	R	S
1	R	R	R	S	S	S	S	S	R	R	R	S
1	R	R	R	S	R	S	S	R	R	R	R	S
1	R	R	R	S	R	S	S	R	R	R	R	R
1	R	R	R	S	R	R	R	S	R	S	R	S
1	R	R	R	S	R	R	R	R	R		R	R
1	R	R	R	R	S	R	R	S	S	R	R	S
1	R	R	R	R	S	R	R	S	R		S	S
1	R	R	R	R	S	R	R	R	S	S	S	R
1	R	R	R	R	S	R	R	R	R	S	R	R
1	R	R	R	R	R	R	R	S	S	R	R	R
1	R	R	R	R	R	R	R	S	R	R	R	S
1	R	R	R	R	R	R	R	R	S	R	S	R
2	R	R	R	R	R	R	R	S	R	R	R	R
1	R	R	R	R	R	R	R	R	S		R	S
1	R	R	R	R	R	R	R	R	R	R	S	S
2	R	R	R	R	R	R	R	R	R	R	S	R
2	R	R	R	R	R	R	R	R	R	R	R	S
8	R	R	R	R	R	R	R	R	R	R	R	R
No. virulent cultures	7	15	21	14	17	17	19	17	19	16	15	22
χ^2 (3:1)[c]	7.56	.24	1.43	.60	.01	.01	.40	.01	.40	.04	.24	2.19
χ^2 (15:1)[c]	2.02											

[a] S = susceptible; R = resistant.

[b] 1 = Ottawa 770B; 2 = Dakota; 3 = Cass; 4 = Abyssinian, Koto, Leona, Ward, and Wells; 5 = Bowman, Clay, Grant, and Minnesota sel.; 6 = Polk; 7 = Marshall; 8 = Birio; 9 = B. Golden sel., Kenya, Norman, and Pale Blue Crimped; 10 = Towner; 11 = Argentine sel., Cortland, and Lino 6899 M.A.; and 12 = Burke. All varieties listed were resistant to race 6 and susceptible to race 22.

[c] Ratio refers to ratio of avirulent to virulent cultures. χ^2 at 5 per cent point, 3.84; at 1 per cent point, 6.64.

Norman, and Pale Blue Crimped, and pathogenicity on this varietal group appeared to be loosely linked with that on Towner.

No crossing over was observed between the genes for virulence on Abyssinian, Koto, Leona, Ward, and Wells; on Bowman, Clay, Grant, and Minnesota selection; on B. Golden selection, Kenya, Norman, and Pale Blue Crimped; and on Argentine selection, Cortland, and Lino 6899 M.A. Hence the assumption that a single gene conditions pathogenicity on all the varieties in each of these 4 group satisfactorily explains the results. In the study of race 22 × race 24 (4), however, pathogenicity on Akmolinsk, susceptible to all cultures of race 6 × race 22 (Hybrid A), was inherited as a unit with pathogenicity on Abyssinian and Leona. Since Abyssinian, Akmolinsk, Koto, Leona, and Ward are differentiated from each other by North American races, they possess different rust-conditioning genes. Consequently, the assumption that the genes for pathogenicity on these varieties are so closely linked that they are inherited as a unit appears to be the more simple explanation. Wells, derived from Pale Verbena (C. I. 416-3), has had the reaction of Leona to all races. These 2 varieties may have an identical gene for rust reaction.

Bowman, Clay, Grant, and Minnesota selection are differentiated by North American races. All of these varieties have not been studied genetically, however, and there may be some duplication of genes. For example, Clay is susceptible and Bowman is resistant to race 7, but these 2 varieties react alike to many races. The possibility that the resistance of Bowman to race 7 is due to a second gene effective against race 7 but not effective against most other North American races has not been determined.

Myers (10) reported 2 genes conditioning rust reaction in Minnesota selection, C. I. 438. One gene conditioned immunity and was allelic to the *L* gene in Ottawa 770B. The second gene conditioned resistance and was allelic to the *M* gene in Newland. Barnes (C. I. 1190), a variety carrying an *L* gene, has been isolated from Minnesota selection × Bison. As Barnes was susceptible to parent races 6 and 22, the resistance of Minnesota selection to certain F_2 cultures of this cross was conditioned by the *M* gene only. Consequently, the F_2 cultures did not differentiate lines carrying both the *L* and the *M* genes of Minnesota selection from lines carrying only the *M* gene.

Bolley Golden selection, Kenya, Norman, and Pale Blue Crimped are differentiated by North American races. The uniqueness of the genes in Bolley Golden selection and Norman has not been established.

Argentine selection, Cortland, and Lino 6899 M.A. have reacted alike to all North American and hybrid races. Although Straib (12) separated European races by the differential reactions of Argentine selection and Lino 6899 M.A., these varieties may possess the same major rust-conditioning gene. Cortland possesses 1 of the genes conditioning rust reaction in Pale Verbena.

Genetics of host-parasite interaction.—The genes for rust reaction in the host are identified by the pathogenicity of specific races of the parasite. And conversely, the genes for pathogenicity in the parasite are identified by the reaction of specific varieties of the host. Varieties having the same reaction to all races are considered to possess identical genes for rust reaction. Races having the same pathogenicity on a variety are considered to possess identical genes for pathogenicity on that variety. The identity of genes for rust reaction in different varieties is provisional only. In these studies, most of the genes for virulence on the differential varieties were derived from race 22. If the genes for resistance to North American races in 2 varieties were allelic and the genes for virulence on these varieties were closely linked in race 22, the varieties would be considered to have identical genes for rust reaction.

Genes for rust reaction in flax have been designated by the symbols *L*, *M*, and *N*, and members of allelic series by superscripts (5, 10). All genes designated by the symbols *L* or *M* have been allelic, but crossing over between some of the genes designated by the symbol *N* has indicated the occurrence of 2 series of linked alleles in the chromosome carrying the *N* genes (5). The relation of some of the *N* genes has been determined. The symbol *N* has been retained for those allelic to the rust-conditioning gene in Bombay. The symbol *P* has been assigned to the genes lying at the other locus. Rust reaction in Clay is conditioned by an independently inherited gene to which the symbol *K* has been assigned. The relation and uniqueness of some genes have not yet been established.

Formerly, the symbols *A* and *V* were used to designate genes for avirulence and virulence, respectively, in the rust fungus, with subscripts indicating the differential variety on which the gene conditioned pathogenicity (4). A new system of gene designation now has been devised to show the specificity of interaction of the genes in host and parasite. The symbol of the gene in flax with which the gene in the rust fungus interacts is used as the subscript to *A* and *V* to indicate this specific relationship. In Table 2 is given the genotype, if established, of the new flax rust differentials, each possessing a single rust-conditioning gene, and the old and new genotype suggested for race 6.

Stakman et al (11) noted the desirability of using the biotype as the basis for concepts regarding races. The gene-for-gene relationship between reaction in the host and pathogenicity in the parasite makes it possible to approach more closely the biotype as the basis for race. A routine test on differentials with single

TABLE 2.—*Probable flax and rust genotypes for a new set of differential varieties in relation to race 6*

Differential variety	C.I. number	Flax genotype[a]				Rust genotype race 6	
						New	Old
Ottawa 770B	355	kk	LL	mm	np/np[b]	A_L A_L	A_o A_o
Dakota	1071	kk	ll	MM	np/np	A_M A_M	A_n A_n
Bombay	42	kk	ll	mm	Np/Np	A_N A_N	A_{bom} A_{bom}
Stewart	1072	kk	L^2L^2	mm	np/np	$A_L{}^2$ $A_L{}^2$	A_{jws} A_{jws}
Cass	1182	kk	ll	M^3M^3	np/np	$A_M{}^3$ $A_M{}^3$	A_{bg} A_{bg}
Koto	842	kk	ll	mm	nP/nP	A_P A_P	$A_{tpb}{}^1$ $A_{tpb}{}^1$
Clay	1188	KK	ll	mm	np/np	A_K A_K	$A_{ar}{}^1$ $A_{ar}{}^1$
Polk	1191	kk	ll	mm	N^1p/N^1p	$A_N{}^1$ $A_N{}^1$	A_{tpb} A_{tpb}
Birio	1085	kk	L^6L^6	mm	np/np	$A_L{}^6$ $A_L{}^6$	A_{ar} A_{ar}
Kenya	709	kk	L^4L^4	mm	np/np	$A_L{}^4$ $A_L{}^4$	A_k A_k
Akmolinsk	515	kk	ll	mm	nP^1/nP^1	$A_P{}^1$ $a_L{}^1$	A_{ak} A_{ak}
Abyssinian	701	kk	ll	mm	nP^2/nP^2	$A_P{}^2$ $A_P{}^2$	A_{ab} A_{ab}
Leona	836	kk	ll	mm	nP^3/nP^3	$A_P{}^3$ $A_P{}^3$	A_{le} A_{le}
Wilden	1193	kk	L^5L^5	mm	np/np	$a_L{}^5$ $a_L{}^5$	
Williston Brown	803	kk	ll	M^1M^1	np/np	$V_M{}^1$ $V_M{}^1$	
Victory A	1170	kk	ll	M^4M^4	np/np	$A_M{}^4$ $a_M{}^4$	
Bowman	1184				[c]	A A	
Bison	389	kk	L^9L^9	mm	np/np	$a_L{}^9$ $a_L{}^9$	
Burke	1180	kk	L^1L^1	mm	np/np	$A_L{}^1$ $A_L{}^1$	A_{bu} A_{bu}
Ward	1181	kk	ll	M^2M^2	np/np	$A_M{}^2$ $A_M{}^2$	$A_{bu}{}^1$ $A_{bu}{}^1$
Pale Blue Crimped	647	kk	L^3L^3	mm	np/np	$A_L{}^3$ $A_L{}^3$	A_P A_P
Norman	1186				[c]	A A	A_{1r} A_{1r}
Bolley Golden sel.	1183	kk	$L^{10}L^{10}$	mm	np/np	A_{L10} A_{L10}	
Grant	1194				[c]	A A	A_{tpb} A_{tpb}
Marshall	1335	kk	ll	mm	N^2p/N^2p	$A_N{}^2$ $A_N{}^2$	
Argentine sel.	705-1				[c]	A A	
Lino M.A. 6899	868				[c]	A A	
Cortland	1512	kk	ll	M^5M^5	np/np	$A_M{}^5$ $A_M{}^5$	A_{le} A_{le}
Wells	1513	kk	ll	mm	nP^3/nP^3	$A_P{}^3$ $A_P{}^3$	
Minnesota sel.	438		L^7L^7		[c]	A A	
Barnes	1190	kk	L^7L^7	mm	np/np	$a_L{}^7$ $a_L{}^7$	
Towner	1561	kk	L^8L^8	mm	np/np	$A_L{}^8$ $A_L{}^8$	

[a] Most of the new differential varieties were derived from hybrids of Bison with the old differentials. As Bison has been susceptible to all North American races, the presence or absence of the Bison gene L^9 in the new differentials with *K*, *M*, *N*, *P*, and not placed rust-conditioning genes has not been determined.

[b] Kerr (9) reports an additional gene in the N chromosome conditioning resistance to certain Australian races.

[c] Genotype not determined.

TABLE 3.—*Utilizing races to identify hybrid plants carrying specific rust-conditioning genes*

Race no.	Race genotype			Reaction[a] of plants possessing genes							
				llmmnn	L	M^3	N^1	LM^3	LN^1	M^3N^1	LM^3N^1
108	a_La_L	$A_M{}^3$	$A_N{}^1$	S	S	R	R	R	R	R	R
123	A_L	$a_M{}^3a_M{}^3$	$A_N{}^1$	S	R	S	R	R	R	R	R
52	A_L	$A_M{}^3$	$a_N{}^1a_N{}^1$	S	R	R	S	R	R	R	R
156	a_La_L	$A_M{}^3$	$a_N{}^1a_N{}^1$	S	S	R	S	R	S	R	R
192	A_L	$a_M{}^3a_M{}^3$	$a_N{}^1a_N{}^1$	S	R	S	S	R	R	S	R
154	a_La_L	$a_M{}^3a_M{}^3$	$A_N{}^1$	S	S	S	R	S	R	R	R

[a] S = susceptible; R = resistant.

rust-conditioning genes identifies the pairs of recessive genes in a rust culture. The homozygosity or heterozygosity of the dominant genes is established by selfing.

Maintenance of varietal resistance. — Rust-resistant varieties of cereals and flax often are damaged by new races or by changes in the prevalence of races of the rust fungi that parasitize them. Jensen (8) proposed several methods of varietal diversification as a means to maintain or prolong the usefulness of a variety. Borlaug (2) proposed the use of the backcross method to develop composite varieties of phenotypically similar lines that are genotypically different for resistance.

In 1947, a backcross program was started to develop lines of flax essentially alike except for a single rust-conditioning gene. The variety Bison was selected as the recurrent parent. Except for its susceptibility to North American races of the flax rust fungus, Bison is well adapted for growing in the North Central States. Lines pure for each rust-conditioning gene have been developed from the Bison backcrosses. As 15 of the 32 varieties to which Bison is being backcrossed possess satisfactory resistance to North American races of the flax rust fungus, a wide choice of rust-resistant Bison-like varieties is available. With an ample supply of resistant germ plasm there appears to be no need at the present time to develop composite varieties of flax as Borlaug (2) has suggested for wheat. Should the need arise, however, the lines derived from the Bison backcrosses should serve this purpose.

Although still ample, the reserve of germ plasm for resistance to North American races of the flax rust fungus has been decreasing. During the decade following 1940, races attacking Koto, Victory A, and Newland were discovered. Whether the races attacking these varieties originated as mutations or had merely escaped detection has not been established. Mutations for virulence are less likely to become established on varieties carrying 2 or more genes than on varieties carrying a single gene for rust reaction. Usually virulence is recessive. Therefore, mutant races can attack heretofore resistant varieties only if they possess, as homozygous recessives, the genes for pathogenicity that complement each gene for resistance in a variety. A race may become homozygous for a mutant gene if the identical mutation occurs in both dikaryotic nuclei of a single spore or if haploids carrying an identical mutation unite through sexual reproduction. In either case, the chance of securing 2 or 3 pairs of mutant recessive genes is much less than that of securing 1 pair.

Bison-like varieties possessing nearly any desired combination of genes for rust reaction within the limits of allelism, may be developed by utilizing the selective pathogenicity of natural and hybrid races to identify the resistant genes in the progeny of hybrids of the Bison backcrosses. For example, the F_1 of the seventh backcrosses on Bison of Ottawa 770B, Cass, and Polk carrying the *L*, M^3 and N^1 genes, respectively, have been intercrossed. Each of these genes is independently inherited and conditions high resistance to all known North American races of the flax rust fungus. As shown in Table 3, the F_1 plants that were resistant to races 108, 123, and 52, each of which attacked 1 parent but not the other 2, possessed a gene for resistance from each parent. The F_1 plants resistant to races 108, 123, and 52 were intercrossed and the progeny inoculated successively with races 156, 192, and 154, each of which attacked plants having a different combination of 2 of the parental genes but not the third. Plants resistant to race 192 possessed the *L* gene, plants resistant to race 156 possessed the M^3 gene, and plants resistant to race 154 possessed the N^1 gene. Plants resistant to races 156 and 192 possessed the LM^3 genes, plants resistant to races 192 and 154 possessed the LN^1, and plants resistant to races 156 and 154 possessed the M^3N^1 genes. Plants resistant to all 3 races possessed all 3 genes for resistance. Plants susceptible to all 3 races possessed none.

DISCUSSION.—The criterion for degree of rust resistance or susceptibility is the pustule type that develops on the host as a result of infection by a race of the parasite. The criterion for degree of avirulence or virulence of a race of the rust fungus is the pustule type that develops on the host as the result of infection by the parasite. Thus, a single phenomenon is the measure of both pathogenicity of the parasite and reaction of the host. Pustule type is the expression of the interaction of the genic and cytoplasmic complex of the host with the genic and cytoplasmic complex of the parasite as affected by the environment. The inheritance of avirulence and resistance or of virulence and susceptibility, as indicated by pustule type, has been explained in this and earlier studies (4, 5, 6) as the result of specific genes in the host interacting with specific genes in the parasite. While purely speculative, it seems reasonable to assume that obligate parasites, such as the rust fungi, must have evolved in association with their hosts. Granting this, a simple

explanation for the high degree of specificity in the rust fungi and other obligate parasites is to assume that during their parallel evolution, host and parasite developed complementary genic systems.

In addition to facilitating studies on the inheritance of pathogenicity, the development of resistant varieties, and the maintenance of varietal resistance, the use of isogenic lines and differential varieties with single rust-conditioning genes may aid in the solution of other problems concerning host-parasite relations.

Few instances of mutations for pathogenicity in the rust fungi have been reported. This is not surprising when the techniques employed in physiologic race studies and the genetics of host-pathogen interaction are considered. To lessen the possibilities of contamination, uredial cultures often are increased on a variety that differentiates them. As avirulence usually is dominant, a mutant less virulent than the parent is unable to infect that differential variety and is lost. Except when genes for pathogenicity are heterozygous, a mutation toward greater virulence (recessive) can be detected only if simultaneous mutations of identical genes occur in both dikaryotic nuclei of a single spore. Even then, the mutation can be detected only if the differential used possesses a single gene for reaction to that race.

For example, an F_1 culture of the cross of race 42 of the flax rust fungus, which attacks all differential varieties except Bombay (13), with race 43, which attacks Bombay only (14), would be heterozygous for all the known genes for pathogenicity. The uredial F_1 culture could be propagated indefinitely as a clonal line and any desired quantity of spores produced. The mutation of only 1 dominant gene to its recessive allelomorph would make that spore homozygous for that pair of recessive (virulent) genes and enable it to attack the differential variety whose resistance was conditioned by the complementary gene in the host. By standardizing inoculation techniques, it should be possible to determine the mutation rate of each gene. The plant breeder could then use those flax genes for rust reaction whose complementary rust genes for pathogenicity showed greatest stability.

Resistance to the rust fungi usually has been inherited as a dominant character, and in flax it has been invariably so. Consequently, a test of a variety with a race that attacks only plants having the dominant gene conditioning reaction in that variety would reveal resistant mutants. These may be increased, homozygous lines secured, and the mutant gene identified by the selective pathogenicity of races of known pathogenic genotype. Mutations for loss of resistance may be more difficult to detect. A plant derived from a resistant variety becomes susceptible to a race avirulent on that variety only if both dominant genes conditioning rust reaction mutate to the recessive allelomorphs. Unless the mutation rate is high, a test of the homozygosity of X_3 populations to detect single-gene mutants is impractical.

Epidemiology studies have greater meaning and utility if the pathogenic genotype of each isolate is known. Since the new flax rust differentials contain both the genes used in developing resistant varieties and those conditioning resistance in commercial varieties, routine pathological tests inform the breeder of changes in the virulence of races affecting his sources of rust resistance and establishes the pathogenicity of each rust isolate on all commercial flax varieties. Each gene for virulence may be identified, and the screening effect of varieties on the prevalence of genes may be determined.

No satisfactory explanation for physiological or hypersensitive resistance to the rust fungi has been given. Allen (1) has suggested that the physiological bases of resistance and susceptibility will benefit greatly from a rigid control of the genotype of both host and parasite. Flor (6) has noted some of the advantages of using lines developed by the backcross method to study the nature of physiological resistance. These lines are essentially alike except for a single rust-conditioning gene. Nutritional differences between such lines or antigen-antibody reactions between such lines and races of the parasite would more likely be related to rust reaction than similar differences between lines that differ in a large number of genes in addition to the gene for rust reaction.

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