RESISTANCE IN MAIZE TO THE TROPICAL AMERICAN RUST FUNGUS, *PUCCINIA POLYSORA* UNDERW.I. GENES Rpp_1 AND Rpp_2

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THE tropical maize rust fungus, *Puccinia polysora* Underw., on its arrival in West Africa in 1949 (Rhind, Waterston and Deighton, 1952) and in East Africa in 1952 (Nattrass, 1953), encountered a susceptible maize population and caused severe losses. Because Cummins (1941) had shown that this species is widespread and long-established in Central America, it seemed possible that resistant maize might have evolved there; and consequently we instituted a search for resistance genes in maize types imported from this source.

The work here described started late in 1952 as a contribution to a co-operative plant-breeding programme for controlling the disease, which has, subject to certain reservations, proved successful (Storey, Howland, Hemingway, Jameson, Baldwin, Thorpe and Dixon, *in press*). Resistance tests of seedlings in the Muguga glass-houses, and breeding from selected plants, had allowed us in 1953 not only to issue to the breeders maize lines carrying resistance genes but also to provide a broad outline of the genetic basis of resistance to guide their work. We continued breeding to complete the genetic analysis; and, not unexpectedly, this has led to some modification of our early conclusions (Storey and Ryland, 1954).

In planning this research and interpreting the data, we received much help from discussions with Sir Joseph Hutchinson, F.R.S., and other colleagues. Experimental methods were guided at the start by the extensive work of pathologists and breeders on the rusts of small cereals and particularly by Dr R. M. Nattrass's experience with the rusts of wheat in East Africa.

1. EXPERIMENTAL METHODS

The general procedure was to test seedlings by inoculation with the rust fungus, to select resistant ones and to grow these to maturity and self-pollinate or cross. All stages were carried out in glasshouses, where work could continue throughout the year without dependence on season. The glasshouses were not temperature-controlled, except that heaters operated when temperatures fell below 70° F. Day length at Muguga is nearly uniform throughout the year. Technical details are as follows.

Plant culture. Seedlings were raised in soil blocks and those selected in tests transplanted to 4-gallon containers and grown on in a large glasshouse. Throughout

growth plants were watered with a dilute nutrient containing: N, 120/p.p.m. as ammonium sulphate; P_2O_5 , 25/p.p.m. as superphosphate, and K, 15/p.p.m. as potassium sulphate. Insect infestations were controlled, without damage to the plants, by treating the soil with Schradan (55 to 66 per cent. A.I.) * at 3 ml. per container, at 6-weekly intervals if needed.

Inoculation. Second and third leaves of young seedlings were painted with 0.25 per cent. gelatine solution and fresh uredospores spread by spatula on the wet surface; or, in later work, a suspension of spores in the gelatine solution was spread by brush.

Infection. Immediately after inoculation, the group of seedlings was covered with a polythene † "hood" for 24 to 48 hours in a shaded glasshouse. (Infection often failed if direct sunlight fell on the plants.) For critical work with rust strains, each seedling was individually covered with a small polythene bag held open by an expanding metal collar pressed into the soil. The collars were heat-sterilised and only new polythene tube was used.

Incubation. After 24-48 hrs. seedlings were transferred to an unshaded or partly shaded glasshouse and hoods removed. Uredosori of the fungus began to appear on inoculated leaves of susceptible plants at 8-10 days from inoculation; and rust reactions were classified at 10, 14 and 18 days.

2. EXPERIMENTAL MATERIAL

The fungus. Two physiologic strains of *P. polysora* were available for this work:—

Race EA.1. Established in our glasshouses from uredospore collections made in 1952 in Zanzibar, Tanganyika and Uganda. At first these "local strains" were maintained in separate houses; and all early tests were replicated in two, and usually all three houses. No significant differences were observed in parallel tests and it is believed that this group constitutes a single physiologic race.

Race EA.2. Appeared spontaneously in the Muguga glasshouses in early 1955 and was recognised by a differential reaction in certain maize lines (Ryland and Storey, 1955). This race has not yet been observed in the field.

The two races have been maintained on maize in separate glasshouses, specially equipped to avoid accidental contamination, by inoculating simultaneously a differential host and a susceptible host. This differential was resistant to Race EA.1, and consequently, if it remained free from sori, the parallel susceptible plants were assumed to be carrying only this race; but the differential was susceptible to Race EA.2 and sori formed on it were necessarily this race.

The plants. We included in our tests 68 maize varieties and lines from African sources and 203 from Central and North America and the Caribbean area. Resistant plants were identified in 45 of the American lines; but the studies here reported are confined to three only, referred to under their "AFRO" accession numbers.

AFRO.24. Received from Dr W. R. Stanton, West African Maize Research Unit, Ibadan, Nigeria, as a first selected progeny from line "SLP 20-4A", from the Rockefeller Foundation, Mexico City.

* As "Pestox III", supplied by Pest Control Ltd.

† "Alkathene" tubular film, supplied by Imperial Chemical Industries Ltd., grade 500 for the large hoods and grade 150 for the small tubes.

AFRO.27. Received from Dr Stanton as a first selected progeny from the cross, "LII 123-2-1-8 × Coah. 8-56A-4-2", from the Rockefeller Foundation, Mexico City.

AFRO.29. Variety "Colombia 2", received from Dr L. M. Roberts, The Rockefeller Foundation, Estacion Agricola Experimental, Medellin, Colombia.

Six susceptible African varieties were used from time to time as parents in crossing with resistant plants, and 8 as experimental controls.

3. THE CLASSIFICATION OF INFECTION TYPES

The following system of qualitative classification, adapted from that of the workers with stem rust of wheat (*e.g.* Stakman, Levine and Loegering, 1944), has been used.

Class "O". No visible effect on the inoculated leaf.

Class "O₁". Visible chlorotic or necrotic lesions on the inoculated leaf, never developing uredosori during the observation period (plate, figs. 1 and 2).

Class "1". Lesions starting as in Class "O₁" but necrotic early, enlarging and developing few or many small uredosori. Frequency of sori indicated by appended signs from "—" to "++" (plate, fig. 3).

Class "4". Uredosori, beginning to open at 8-10 days, forming spores freely, later enlarging and sometimes expanding by rings of secondary sori. Necrosis absent in early stages; chlorosis variable but typically no more than a barely visible paling of the leaf tissue, rarely a yellow diffuse "halo" around the sori (plate, figs. 5 and 6).

Class "X". Intermediate between Classes "1" and "4". Typically Class "1" lesions with small sori, mixed with larger sori on intervening green tissue (plate, fig. 4).

Class "O₁", although based on the objective character of absence of sori, proved to be in part an unfortunate choice, for experience showed that two grades could be recognised. In Grade "(a)" the individual lesions were $\frac{1}{4}$ to $\frac{1}{2}$ mm. diameter, normally diffuse-edged and chlorotic (plate, fig. 1) but sometimes becoming necrotic and brown. In grade "(b)" lesions expanded to 1 mm. and often more in diameter and became necrotic early (plate, fig. 2). Grade "(a)" is normally distinctive; Grade "(b)" differs in character from Class "1" only in lacking sori, and appears to be one end of a series passing through "1—" to "1++".

In its typical form Class "X" is distinctive; but we have been forced to use it for a range of intermediates between "1++" and "4". At the ends of this range differentiation has been difficult and uncertain.

4. RESULTS OF EXPERIMENTS

Early experiments were necessarily confined to inoculating maize with rust Race EA.1; and the reaction to this race was the criterion of selection for all the breeding described. Only after EA.2 had been isolated in 1955 was it possible to test sample maize lines against this race.

(i) *The susceptible reaction*

Sixty-eight African maize varieties, many of which were known to be susceptible in the field, reacted Class "4" in tests against EA.1. A sample of 15 of these, tested against EA.2, also reacted "4". These variety tests included 1094 seedlings inoculated with EA.1 and 180 with EA.2. In addition, as experimental controls some ten thousand seedlings of East African varieties were inoculated with EA.1 and one thousand with EA.2. The only deviations from the characteristic Class "4" reactions were :—(1) a number classed "O", due, we believe, to accidental failure of the inoculation (see below) ; and (2) a number showing exceptionally severe chlorosis.

Seven Class "4" plants, selected from families of American origin that had segregated into "O₁", "1" and "4", gave on selfing only Class "4" plants in 93 seedlings of the S₁ families.

We conclude that a Class "4" plant does not carry a gene for resistance of the kind here studied. We are less certain whether the severely chlorotic "4" reaction may be genetically controlled. There are strong indications that chlorosis is often determined by the plant's nutrition and environment ; and experimental tests of families derived from chlorotic parents, under the limited environmental control available to us, gave no decisive evidence that chlorosis was inherited. In certain American maizes, however, we encountered a characteristic chlorotic reaction that is inherited. Our studies of this will be reported later.

(ii) *The resistant reactions*

Workers with wheat rusts interpret Class "O" as denoting immunity. We have not found this to be true of maize and *P. polysora*. Thus 16 families were raised by selfing Class "O" plants recorded in families otherwise susceptible ; all of 195 plants in the S₁ families were classed "4". Only when Class "O" plants were selected from families segregating "O₁", "1" and "4" was there any exception ; two such S₁ families themselves segregated "O₁", "1" and "4". We conclude that Class "O" in our experiments has denoted no more than accidental escape from infection ; and indeed, when methods had been improved by experience, very few plants were ever recorded "O" on both inoculated leaves.

For this reason we have excluded Class "O" plants from the records of our experiments.

The form of resistance that we have studied is that denoted by Classes "O₁", "1" and "X". In accordance with current thought on the rust fungi, we interpret these classes as indicating a hypersensitive reaction by the plant, causing the invading mycelium to be starved of nutrients and so prevented from developing to the sporing stage or hindered from producing more than small sori. We briefly reported our conclusion that this hypersensitive reaction is controlled by a single dominant major gene (Storey and Ryland, 1954) ; but

we now modify this by postulating two such genes as recognisable under conditions existing in East Africa to-day.

Resistance in AFRO.29. Seedlings of this variety, as received, reacted to *P. polysora*, Race EA.1, as follows :—

$$9\text{—“O}_1\text{”} : 5\text{—“1”} : 32\text{—“4”}.$$

In table 1 are assembled the results obtained in testing families derived from selected “O₁” and “1” plants. They are grouped in two main series, “I” and “II”, with sub-series “A” for selfed progenies and “B” for crosses with susceptibles.

In tests of Series I against rust Race EA.1 the S₁ families from two selected “O₁”s segregated into “O₁” and “4” in proportions not significantly different from 3 : 1 ($P = 0.1$). Selected “O₁”s from these families gave 4 S₂ families that were all “O₁”; and, since 3 S₃ families from these were also classed “O₁” only, the families are considered to be homozygous for a resistance gene. Five S₂ families segregated 3 : 1.

When “O₁” plants were crossed with susceptibles (Series IB), 3 F₁ families segregated into 1 “O₁” : 1 “4”. The F₂, by selfing “O₁”s from the F₁, segregated 3 : 1 in all of 10 families. The F₃ showed 8 families all classed “O₁” and 14 that segregated.

In all these tests the resistant class was “O₁”, Grade “(a)” ; except that 3 Class “1” seedlings were recorded in a total of 758 resistant ones. The “O₁”s varied within a narrow range, mainly showing only chlorotic lesions but, less frequently, similar sized lesions that were brown and necrotic. There was no evidence that homozygous plants were consistently different from those that were heterozygous. Our procedure, because our main purpose was breeding for practical control, was to select as parents for later generations those plants showing only the mildest chlorotic reaction ; nevertheless, the proportions of 4 homozygous to 5 segregating families in the S₂ generation, and 8 to 14 in the F₃, are not significantly different from the 1 : 2 ratio expected from a random selection of resistant parents within a family segregating 3 : 1.

When sample families from the above lines were tested against Race EA.2 they were recorded Class “4” ; their reactions were characteristic of susceptible controls, although there was a tendency for the sori to be smaller at first and to expand more slowly. One exception, which we cannot explain, was encountered in 2 F₃ families from the cross. The F₂ family from which both arose had segregated normally into “O₁” and “4” against EA.1 ; but both F₃ families were recorded “O₁” against EA.1 and severe “1” against EA.2.

The evidence of Series I, on balance, therefore, indicates a single, fully dominant gene, conferring the power to react “O₁ (a)” against Race EA.1 but failing to confer any resistance against EA.2. If the gene is subject to modification by minor genes—accounting possibly

TABLE 1
Tests of derivatives from AFRO.29 by (A) selfings, (B) crossing with susceptibles

Series	Gener- ation	Group	Origin and rust classes of parents against EA.1	Totals in each rust class in tests against EA.1				Totals in each rust class in tests against EA.2			
				No. of families tested	Resistant		Segregation ratio postulated*	No. of families tested	Resistant		Susceptible
					"O ₁ "	"1"			"O ₁ "	"1"	
IA	S ₁ S ₂ S ₃	(a) (b)	AFRO.29 ("O ₁ ") selfed	2	41	0	3:1	...	...	...	...
			Selected S ₁ ("O ₁ ") selfed	4	160	0	1:0	3	0	0	31
			Same	5	74	1	3:1	...	...	...	...
			Selected S ₂ (a) ("O ₁ ") selfed	3	42	0	1:0	1	0	0	8
IB	F ₁ F ₂ F ₃	(a) (b)	AFRO.29 ("O ₁ ") × susceptible ("4") and reciprocal	3	24	0	1:1	3	0	0	38
			Selected F ₁ ("O ₁ ") selfed	10	108	0	3:1	2	0	0	20
			Selected F ₂ ("O ₁ ") selfed	8	173	0	1:0	6	0	0	60
			Same	14	133	2	3:1	2	0	0	14
IIA	S ₁ S ₂	(a) (b)	AFRO.29 ("1") selfed	1	0	29	3:1	1	0	3	3
			Selected S ₁ ("1") selfed	1	1	11	1:0	1	3	8	0
			Same	1	1	5	3:1	1	0	3	2
			Susceptible ("4") × AFRO.29 ("O ₁ ")	1	8	0	1:1	1	0	5	5
IIB	F ₂ F ₃		Selected F ₁ ("O ₁ ") selfed	3	29	0	3:1	2	9	14	3
			Selected F ₂ ("O ₁ ") selfed	3	21	0	3:1	1	2	8	4

* Ratio expressed as—(Resistant Classes) : (Susceptible Class). The χ^2 tests have revealed no significant deviation from these ratios, either in the totals recorded above, or—with rare exceptions—in the individual numbers for each separate family. This is true also of all later tables where segregation ratios have been postulated.

for the variation in intensity of necrosis—this effect has been only slight in our trials. We designate this gene **Rpp₁**.*

Series II of table 1 is markedly different. The parent of the selfed lines was itself classed “1” against EA.1, and the resistant plants in its derivatives were predominantly “1”, both against EA.1 and also against EA.2. The same held generally for families derived from crosses with susceptibles, except that the resistant plants were all classed “O₁” against EA.1 (but mostly “1” against EA.2). Family ratios did not deviate significantly from those expected if a single dominant gene was concerned; but this gene is not **Rpp₁** because it is effective against Race EA.2. The gene is indistinguishable, on the limited evidence of this series of AFRO.29 derivatives, from the **Rpp₂** identified in AFRO.24.

Resistance in AFRO.24. The original AFRO.24 material proved on test to be all resistant to Race EA.1, reactions being classed “O₁” and “1”. Figures from testing derivatives from (A) selfs and (B) crosses are presented in table 2, in the same general form as that used in table 1.

In the selfed lines, one S₁ family was entirely resistant to EA.1 (Classes “O₁” and “1”), and 3 S₂ families in this line were also resistant. Nine S₁ families segregated 3 resistants to 1 susceptible; and, of the S₂ families raised from selected resistants from the segregating S₁ families, 6 were homozygous and 6 segregated. Four of the homozygous families, tested against Race EA.2, were entirely resistant (“O₁” and “1”).

The derivatives from crosses with susceptibles behaved generally in accordance with expectation. One F₁ family contained only resistant plants, and the other segregated 1 : 1. All the 17 F₂ families segregated 3 : 1. Of the F₃ families, 23 were presumed homozygous and 8 segregated. Six of the homozygous families, tested against EA.2, were recorded “O₁” and “1” only.

The gene from AFRO.24 differs from the **Rpp₁** of AFRO.29, not only in conferring equal resistance to the two rust races, but in other respects also. Whereas plants carrying **Rpp₁** reacted against EA.1 in the Class “O₁ (a)” with minor variations, derivatives of AFRO.24 might show the whole range from “O₁ (b)” to “1++”. This wide range can be attributed in part, but only in part, to environmental differences encountered in experiments done at different seasons. To eliminate this environmental effect, and to obtain more exact figures for the several grades of Class “1”, we repeated tests of certain families, against EA.1 only, all inoculations being done during 5 days in June 1956 (table 3). On this occasion homozygous lines in the S₁ and S₂ showed only “O₁ (b)” phenotypes; while segregating selfed lines showed the range from “O₁ (b)” to “1+” and “4”. Among the crosses, two homozygous F₃ families (last line of the

* This designation has been adopted on the advice of Dr M. M. Rhoades, Secretary to the Maize Genetics Co-operation, the University of Illinois, U.S.A.

TABLE 2
Tests of derivatives from AFRO.24, by (A) selfing, (B) crossing with susceptibles

Series	Generation	Group	Origin and rust classes of parents against EA.1	Totals in each rust class in tests against EA.1				Totals in each rust class in tests against EA.2			
				No. of families tested	Resistant	Susceptible	Segregation ratio postulated*	No. of families tested	Resistant	Susceptible	
					"O ₁ "	"1"			"O ₁ "	"1"	"4"
A	S ₁	(a)	AFRO.24 ("O ₁ ") selfed	1	23	12	1:0	...	...	...	...
	(b)		AFRO.24 ("O ₁ "-"1") selfed	9	53	50	3:1	...	...	...	...
	S ₂	(a)	Selected S ₁ (a) ("O ₁ "-"1") selfed	3	36	24	1:0	2	22	4	0
	(b)		Selected S ₁ (b) ("O ₁ "-"1") selfed	6	117	27	1:0	2	22	3	0
	(c)		Same	6	38	21	3:1	...	...	...	...
B	F ₁	(a)	Susceptible ("O ₁ ") × AFRO.24 ("4")	1	0	35	1:0 (heterozygous)	1	0	6	0
	(b)		Same	1	0	6	1:1	...	...	...	...
	F ₂		Selected F ₁ (a) and (b) ("1") selfed	17	13	140	3:1	...	...	...	...
	(a)		Selected F ₂ ("O ₁ "-"1") selfed	23	70	440	1:0	5	8	24	0
	(b)		Same	8	6	55	3:1	...	...	...	...

* See note on table 1.

table) were all "O₁ (b)" or "1—", whereas known heterozygous resistants in the F₁ were "1+" or "1++". Resistant plants in segregating families in the F₂ and F₃ generations covered the full range from "O₁ (b)" to "1++". We interpret this evidence as indicating that the gene is incompletely dominant.

Support to this hypothesis is given by the proportions of homozygous to segregating families realised in the S₂ generation and the F₃ from crosses (table 2). The 6:6 proportion in the S₂ is not significantly different from the 1:2 ratio expected from random selection of parents in a family segregating 3:1; but 23:8, in the

TABLE 3
Second-series tests of certain families derived from AFRO.24
(Inoculated with EA.1 only in June 1956)

Series	Nature of families	Segregation ratios in first tests	No. of families tested	Totals in each rust class					
				"O ₁ (b)"	"1—"	"1—"	"1+"	"1++"	"4"
A	S ₁ and S ₂ AFRO.24	1:0	2	52	0	0	0	0	0
	Same	3:1	5	10	11	4	5	0	14
B	F ₁ —Suscept. × AFRO.24	1:0 (heterozygous)	1	0	0	0	2	8	0
	Same	1:1	1	0	0	0	0	4	5
	F ₂ and F ₃ from cross	3:1	6	8	13	14	9	8	6
	F ₃ of cross	1:0	2	8	12	0	0	0	0

F₃, is very markedly so. In fact, we had selected as parents those plants showing the least severe reactions—Class "O₁" preferably, or the mildest "1". This selection appears to have resulted in the propagation of a disproportionate number of homozygotes, indicating again that the gene is incompletely dominant.

Nevertheless, it is evidently not possible to select genotypes with certainty by inspection; there is no more than a tendency for the selection we have practised to give homozygotes. It seems necessary to postulate some modification by minor genes, causing the reactions of each genotype often to overlap two or more sub-classes.

We designate the major gene from AFRO.24 as the "type" of Rpp₂.

Resistance in AFRO.27. The original imported material, tested against rust Race EA.1, was recorded:—

1—"O₁" : 12—"1" : 20—"4".

Records from testing selfed and out-crossed derivatives from selected "O₁" and "1" parents are recorded in table 4.

Results obtained are closely similar to those from derivatives of AFRO.24, except that throughout there is a tendency for reactions

TABLE 4
Tests of derivatives from AFRO.27, by (A) selfing, (B) crossing with susceptibles

Series	Gener- ation	Group	Origin and rust classes of parents against EA.1	Totals in each rust class in tests against EA.1				Totals in each rust class in tests against EA.2				
				No. of families tested	Resistant		Susceptible	Segregation ratio postulated*	No. of families tested	Resistant		Susceptible
					"O ₁ "	"1", "X"	"4"			"O ₁ "	"1", "X"	"4"
A	S ₁	(a)	AFRO.27 ("1") selfed	2	28	63	0	1:0	2	6	6	0
	S ₂	(b)	AFRO.27 ("O ₁ " "1") selfed	4	5	35	21	3:1	...	...	...	...
		(a)	Selected S ₁ ("O ₁ " "1") sibbed	1	8	36	0	1:0	1	0	10	0
	(b)	Selected S ₁ (b) ("O ₁ " "1") selfed	5	7	105	0	1:0	5	12	44	0	
	(c)	Same	8	0	97	28	3:1	...	...	...	...	
B	F ₁		AFRO.27 ("1") × susceptible ("4") and reciprocal	2	0	12	24	1:1	...	...	...	...
	F ₂	(a)	Selected F ₁ ("1") selfed	3	0	27	7	3:1	...	...	...	...
	F ₃		Selected F ₂ ("1") selfed	2	0	43	0	1:0	2	9	32	0
		(b)	Same	2	0	14	8	3:1	...	...	...	...

* See note on table 1.

to be more severe. Indeed, the range extended beyond that characteristic of "1++" into the rather indeterminate Class "X"; because, however, our early records are insufficiently detailed, we have grouped the "1" and "X" reactions in table 4.

If we combine "O₁", "1" and "X" as constituting together a resistant group, the segregation ratios realised point to a single dominant, or partly dominant, major gene. In tests against EA.1, 2 S₁ families—and an S₂ from one of these—were wholly resistant; and segregating families in the selfed lines gave proportions not significantly different from a 3:1 ratio. Among the crosses with susceptibles, the F₁ segregated 1:1 (but barely significantly), the F₂ 3:1, and, in the F₃, 2 families were homozygous and 2 segregated.

TABLE 5

*Second-series tests of certain families derived from AFRO.27
(Inoculated with EA.1 only in May 1956)*

Series	Nature of families	Segregation ratios in first tests	No. of families tested	Totals in each rust class			
				"O ₁ "	"1"	"X"	"4"
A	S ₁ of AFRO.27	1:0	2	0	94	2	0
	S ₁ and S ₂ from AFRO.27 . .	3:1	8	0	24	35	14
B	F ₁ —Susceptible "X" AFRO.27	1:1	2	0	0	5	9
	F ₂ and F ₃ —Selfs	3:1	4	0	11	14	11
	F ₃ —Self	1:0	1	0	14	0	0

Ten selected homozygous families were tested also against Race EA.2; all were recorded "O₁" and "1-X".

The proportions of homozygous to segregating families in the S₂ and F₃ generations (7:10, in total) did not indicate that in selecting their parents we had obtained a significant excess of homozygotes. Nevertheless, as with AFRO.24, we repeated tests against EA.1 of certain families during May 1956 (table 5). In this series, Class "O₁" was not encountered. Believed homozygous families were predominantly Class "1" (108 "1"s to 2 "X"s). Two F₁ families from crosses, where the resistant plants were necessarily heterozygous, gave only "X" resistants. In segregating families the resistant plants were distributed in Classes "1" and "X". There is, therefore, a suggestion of incomplete dominance.

Thus in AFRO.27 we appear again to have identified an incompletely dominant gene conferring equal resistance to rust Races EA.1 and EA.2. Although the resistant reaction in homozygotes is not so strong as when Rpp₂ from AFRO.24 is present and the grade of the heterozygote is lower, we do not feel justified, in the absence of more conclusive evidence, in postulating for AFRO.27 a major gene different from Rpp₂.

5. DISCUSSION

It is hardly open to question that the genes we have designated **Rpp₁** and **Rpp₂** are not identical. The primary difference lies in the differential reactions against the two physiologic races of *P. polysora* existing in East Africa: **Rpp₁** confers high resistance against EA.1, but none against EA.2, while **Rpp₂** confers equal resistance against both races. Furthermore the "O₁ (a)" reaction of **Rpp₁** is normally distinguishable by inspection from the "O₁ (b)" of **Rpp₂**, the highest grade of resistance this gene is capable of conferring. **Rpp₁** appears to be completely dominant, and its resistant reactions are only slightly, if at all, modified by minor genes.

On the other hand, **Rpp₂**, as identified in AFRO.24, besides showing no differential reaction against the two rust races, is incompletely dominant, and the range of the resistant reactions seems most reasonably explicable as minor-gene modification.

Whether we are correct in suggesting that the gene obtained from AFRO.27 (and from AFRO.29 in part) is identical with that from AFRO.24 is uncertain. The AFRO.27 gene resembles it in being equally resistant to both races of the rust, in being incompletely dominant and in being apparently subject to considerable minor-gene modification. That the AFRO.27 derivatives show more severe reactions than those from AFRO.24 might be due to differences in the minor-gene complements of these two lines. It is possible that new physiologic races may appear in East Africa against which the genes from these two sources may react differently; but meanwhile the preferable course is to regard them as identical.

The expectation that maize resistant to *P. polysora* would have evolved in the home of this fungus has been realised, as Stanton and Cammack (1953) have reported on other evidence. It is surprising that, of the 203 separate samples of maize received from American sources, not one was homozygous for a resistance gene. At first sight it appears that selection pressure at source has not been as great as might have been expected from the severe effects of the disease in Africa. But it must be remembered that in East Africa we have studied the genes conferring resistance to only two races of *P. polysora*; and it is possible that in Central America maize has evolved under infection by a range of physiologic races largely different from those with which we have worked.

6. SUMMARY

1. When *Puccinia polysora* appeared in Africa, it encountered a susceptible maize population; but resistance of the hypersensitive type was found in certain maize lines introduced from Central America. The genetics of resistance were studied by controlled inoculation of seedlings in glasshouses and by breeding from individual plants selected on seedling tests.

Plate

Portions of leaves from maize seedlings inoculated with *Puccinia polysora* Underw.
All $\times 4.5$.

- FIG. 1.—Class “O₁”, Grade “(a)”, at 18 days. Typical reaction of plant carrying **Rpp₁**; the lesions are yellow, slightly shrunken and remain small.
- FIG. 2.—Class “O₁”, Grade “(b)”, at 18 days. A rather severe form of the reaction in a plant carrying **Rpp₂**. The lesions are necrotic and pale brown. (Only half the leaf was photographed.)
- FIG. 3.—Class “1++”, at 18 days. Typical severe reaction in plant carrying **Rpp₂**. Lesions pale brown, with small open sori.
- FIG. 4.—Class “X”, at 18 days. Lesions similar to those of “1++”, together with some larger sori (mainly on left-hand side of photograph), erupting from leaf tissue only slightly chlorotic.
- FIG. 5.—The susceptible reaction, Class “4”, at 10 days. Sori just erupting from tissue only faintly chlorotic.
- FIG. 6.—Class “4” at 20 days. The sori have enlarged greatly and extruded large masses of uredospores. Chlorosis still confined to faint yellowing of the leaf tissue.

FIG. 1

FIG. 2

FIG. 3

2. Two major genes for resistance were recognised. The first, **Rpp₁**, conferred high hypersensitivity against the prevalent physiologic race of *P. polysora* (Race EA.1), made evident on inoculated leaves by minute chlorotic-necrotic lesions without uredosori; but it conferred no recognisable resistance to a second race of the fungus (EA.2). **Rpp₁** appeared to be fully dominant. The second gene, **Rpp₂**, conferred a lesser hypersensitivity, typically necrotic lesions with small sori, but was equally effective against Races EA.1 and EA.2. This gene was incompletely dominant. **Rpp₂** appeared to be subject to considerable minor-gene modification, but **Rpp₁** much less so.

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