HEREDITY VOLUME 25 PART 3 AUGUST 1970

GENETICAL STUDIES ON RESISTANCE TO POTATO VIRUSES X AND Y

G. COCKERHAM
Scottish Plant Breeding Station, Pentlandfield, Roslin, Midlothian

Received 1.viii.6

1. INTRODUCTION

EXTREME resistance (immunity) to potato virus X and/or potato virus Y and necrotic hypersensitivity, a basis of resistance to these viruses, are found in various species of the tuber-bearing Solanaceae and there is a consensus of evidence that each form of response is under single gene control (Cadman, 1942; Cockerham, 1945, 1958, 1962; Mills, 1965; Ross, 1954*a*, *b*, 1960). Of the genes concerned, some are comprehensive in scope and are brought into action by all strains of the virus to which they are related; others are invoked by only a portion of the strain spectrum of that virus. Conversely, some virus strains evoke the resistant or necrotic response in the presence of any gene, specific or comprehensive, related to the virus; other strains are more closely related to specific genes; and yet other strains evoke only those genes which have comprehensive coverage. These latter relationships form the basis of strain classification adopted by Cockerham (1954) for potato virus X and they also serve to distinguish viruses A and C from the main body of potato virus Y (Cockerham, 1943*b*, 1958).

Relationships also exist between the genes themselves both in linkage (Cockerham, 1945; Howard and Fuller, 1965) and in allelism (Cockerham, 1958; Ross, 1960) and it is with further exploration within this particular field of enquiry that the present investigations are concerned.

2. MATERIALS AND METHODS

Material for study is drawn mainly from species which have been used or explored as sources of virus resistance in potato breeding. The species are *S. chacoense* and *S. microdontum*, (diploids with 2n = 24); *S. acaule*, *S. tuberosum* (British, European and North American cultivars), *S. tuberosum* subspecies *andigena* and *S. stoloniferum* (tetraploids with 2n = 48); and *S. demissum* and *S. hougasii* (hexaploids with 2n = 72). Additionally, the diploid species *S. sparsipilum* and di-haploid seedlings derived from *S. tuberosum* have been used as link material between the diploid and tetraploid species.

Three strains of potato virus X were used, X^L, X^B, and X^4, which evoke respectively the gene Nx and comprehensive genes, the gene Nb and comprehensive genes, and comprehensive genes only (Cockerham, 1954, Cockerham and Davidson, 1963). Their counterparts were potato virus A which evokes specific Na genes as well as genes with comprehensive effect in relation to potato virus Y; potato virus C which is specific for the gene Nc but also evokes comprehensive genes; and a common strain of potato virus Y which, in the context of the material used, evokes comprehensive genes only (Cockerham, 1945, 1958). Each virus was maintained in a cultivar

selected to reduce to a minimum the chances of cross-infection and each was used either directly from the cultivar or after transference to White Burley tobacco or to selected homozygotes of *S. stoloniferum* which were found to furnish excellent scions for graft inoculation.

Infection of experimental material was achieved through graft unions with infected scions; by dusting leaves with either carborundum or celite abrasive and then rubbing them with infective plant sap derived from potato or tobacco leaves; or by spraying, under pressure, young seedlings with infective sap containing abrasive. The method used was determined by the circumstances of the virus concerned, the phenotypic responses expected and the age and condition of the experimental material. Phenotypes were distinguished as resistant, that is, with null response or showing localised micro-necrosis; necrotic, with macro-necrosis often systemic and lethal; and susceptible. The latter were detected not only by the symptoms expressed but also by serological tests for the presence of X viruses and by the use of detached leaves of two sensitive lines of *S. demissum* as test material for viruses A, C and Y (Cockerham, 1958).

3. Genetical interpretation

Much of the data to be presented is derived from cultivars of *Solanum tuberosum* or from hybrids between such cultivars and *Solanum andigena* and *Solanum acaule*. The genes concerned are inherited in tetrasomic fashion and all results are consequently interpreted on the basis of random chromosome association at meiosis. In a few cases there is indication, through the occurrence of occasional double recessive seedlings where none is expected, of double reduction and hence of random chromatid association. These are so infrequent, however, that they are interpreted as exceptional rather than usual.

Furthermore, the inheritance of " immunity " from virus X has been attributed to (1) the operation of a complementary pair of genes (Stevenson *et al.*, 1939) or (2) to a recessive gene of which the dominant counterpart conditions necrotic, hypersensitive reactions in the simplex, duplex and triplex condition and non-necrotic susceptibility in the quadriplex condition (Hutton and Wark, 1952). The present data were examined in the light of both these hypotheses but neither gave adequate fit over the whole range. It now seems clear that single dominant genes control both resistant and necrotic responses but that disturbed segregations due to undetermined causes are not infrequently encountered.

In the cases of diploid and di-haploid material and in *S. stoloniferum*, *S. demissum* and *S. hougasii* inheritance was, in general, disomic and is so interpreted.

4. Results

(A) *Genes of commercial varieties of potatoes* (tables 1, 2 and 3)

Data on the occurrence, distribution, significance and inheritance of four genes, Nx, Nb, Na and Nc, which control hypersensitive response to specific portions of the strain spectrum of virus X (genes Nx and Nb) and virus Y (genes Na and Nc) are given in earlier papers (Black, 1956; Cadman, 1942; Cockerham, 1939, 1943*a*, 1943*b*, 1945, 1952, 1954, 1962). The relationships of the four genes were examined in two progenies of the cross

between Craigs Defiance (Nx, Nb, Na, Nc) and Flourball (nx, nb, na, nc). The results from each progeny and from the two progenies combined are given in table 1.

TABLE 1

Progenies 758a and 758b. Craigs Defiance × Flourball

(i) *Single factor segregations, necrotic (nec.), susceptible (sus.)*

	Progeny (a)				Progeny (b)				Combined (a+b)			
	Observed				Observed				Observed			
Virus	nec	sus	χ^2 1:1	P	nec	sus	χ^2 1:1	P	nec	sus	χ^2 1:1	P
X^L	90	77	1·012	>0·3	58	59	0·008	>0·9	148	136	0·507	>0·3
X^B	73	75	0·027	>0·8	44	48	0·174	>0·5	117	123	0·150	>0·5
A	75	63	1·043	>0·3	54	43	1·247	>0·3	129	106	0·690	>0·3
C	85	75	0·625	>0·3	54	53	0·009	>0·9	139	128	0·453	>0·5

(ii) *Joint segregations, two factors*

Dominant genes in phenotype	Progeny (a) Observed	χ^2 1:1:1:1	P	Progeny (b) Observed	χ^2 1:1:1:1	P	Combined (a+b) Observed	χ^2 1:1:1:1	P
Nx, Nb	40	1·054	>0·7	20	0·652	>0·8	60	0·548	>0·9
Nx, —	39			25			64		
—, Nb	32			24			56		
—, —	36			23			59		
Nx, Na	69	117·33	v. small	47	71·038	v. small	116	182·36	v. small
Nx, —	2			1			3		
—, Na	5			6			11		
—, —	61			42			103		
Nx, Nc	37	57·26	v. small	29	0·867	>0·8	66	2·122	>0·5
Nx, —	45			23			68		
—, Nc	47			25			72		
—, —	28			28			56		
Nb, Na	39	3·584	>0·1	24	1·289	>0·7	63	3·960	>0·2
Nb, —	25			19			44		
—, Na	35			26			61		
—, —	38			21			59		
Nb, Nc	40	0·728	>0·5	20	2·696	>0·3	60	1·385	>0·7
Nb, —	33			24			57		
—, Nc	38			29			67		
—, —	36			19			55		
Na, Nc	35	3·584	>0·1	31	4·609	>0·02	66	2·591	>0·3
Na, —	39			19			59		
—, Nc	38			18			56		
—, —	25			24			49		

(iii) *Joint segregation, three factors (excluding Na)*

Dominant genes in phenotype	Progeny (a) Observed	χ^2	Progeny (b) Observed	χ^2	Combined (a+b) Observed	χ^2
Nx, Nb, Nc	20	1:1:1:1:1:1:1:1	11	1:1:1:1:1:1:1:1	31	1:1:1:1:1:1:1:1
Nx, Nb, —	20		9		29	
Nx, —, Nc	14	9·233	16	4·480	30	3·979
Nx, —, —	24		9		33	
—, Nb, Nc	20		9		29	
—, Nb, —	12	P>0·2	15	P>0·7	27	P>0·7
—, —, Nc	24		13		37	
—, —, —	12		11		23	

The data in section (i) of this table agree well with the earlier findings that top necrosis to each of the viruses concerned is conditioned by single genes which, in the variety Craigs Defiance, are present in the simplex condition. From the joint segregations, (ii), it is clear that Nb segregates independently of Nx, Na and Nc and that Nx and Na are closely linked in the coupling phase. There are discrepancies, however, in the relationships between Nx and Nc and Na and Nc. From progeny (a) it would seem that Nx is possibly linked to Nc with the two genes in the repulsion phase. This is not confirmed in the data of progeny (b). Also, it would seem from

progeny (b) that Na is possibly linked to Nc in the coupling phase, again unconfirmed by the data from the other progeny. No complete explanation of this anomalous behaviour can be given but it is of interest to relate it to the data of Howard and Fuller (1965) who found Nx, Na and Nc were apparently in the same linkage group in the variety Southesk whereas Nx and Nc showed independent inheritance in a progeny from the variety Ulster Knight which itself was bred from Craigs Defiance. In the present case the three factor segregations, (iii), indicate that the overall situation for the genes Nx, Nb and Nc is one of independent inheritance.

Confirmation of the independence of the Nx and Nb genes was obtained from the examination of a progeny 5367 derived from a seedling of the variety Cardinal, which is duplex for the gene Nx (Cadman, 1942), and Catriona,

TABLE 2

Progeny 5367. 4383(3) × Flourball

(i) *Single factor segregations*

Virus	Phenotypes nec	sus	χ^2 5 : 1	χ^2 1 : 1	P
X^L	66	11	0·313	—	>0·5
X^B	42	33	—	1·08	>0·2

(ii) *Joint segregation*

Dominant genes in phenotype	Observed	Expected with Nx, Nb alleles	χ^2	P	Expected with Nx, Nb independent	χ^2	P
Nx, Nb	42	25·67			32·08		
Nx, —	25	38·50	14·699	v. small	32·08	5·443	>0·1
—, Nb	5	12·83	(—, Nb and —, —		6·42		
—, —	6	0	combined)		6·42		

duplex for the gene Nb (Cockerham, 1962), crossed with the variety Flourball which contains neither gene. The results of this examination (table 2) show that the seedling parent was itself duplex for the gene Nx and simplex for Nb. In the event of the two genes being alleles it would be expected that, in the absence of double reduction, there would be no segregates susceptible to both strains of the virus. There were, however, six seedlings of this type and the data fit to the 5 : 5 : 1 : 1 expectation indicative of independent inheritance of the two genes.

Data on linkage between the genes Nx and Na, as indicated in table 1, are summarised in table 3 from which it is seen that within each of the varieties Craigs Defiance, Liddesdale Lads, Kepplestone Kidney, Edgecote Purple and Cardinal, linkage occurs in the coupling phase. Recombination values, estimated from the simplex backcrosses only, appears to vary between varieties and between their usage as male or female parents. There is, however, resaonable consistency between the values obtained on the female side and a mean estimate of $5{\cdot}43 \pm 0{\cdot}80$ per cent. is obtained from the eight progenies available for calculation.

The behaviour of Southesk is anomalous and inexplicable in that there

TABLE 3

Linkage between Nx and Na

Parents	Genetic type	No. progenies	Single factor segregation: Virus X nec	Single factor segregation: Virus X sus	Single factor segregation: Virus A nec	Single factor segregation: Virus A sus	Joint segregation X/A reactions: nec/nec	nec/sus	sus/nec	sus/sus	Recombination %
Southesk × recessive	$Nx(nx)_3.Na.(na)_3 \times (nx)_4(na)_4$	3	169	210	171	208	84	85	87	123	—
Craigs Defiance × recessive	$Nx(nx)_3.Na.(na)_3 \times (nx)_4(na)_4$	3	155	152	163	144	150	5	13	139	5·86
Liddesdale Lads × recessive	$Nx(nx)_3.Na.(na)_3 \times (nx)_4(na)_4$	2	90	89	89	90	85	5	4	85	5·30
recessive × Liddesdale Lads	$(nx)_4(na)_4 \times Nx(nx)_3.Na(na)_3$	1	81	85	81	85	76	5	5	80	6·03
Kepplestone Kidney × recessive	$Nx(nx)_3.Na(na)_3 \times (nx)_4(na)_4$	2	85	90	82	93	78	7	4	86	6·29
recessive × Kepplestone Kidney	$(nx)_4(na)_4 \times Nx(nx)_3.Na(na)_3$	2	126	93	126	93	124	2	2	91	1·83
Edgecote Purple × recessive	$Nx(nx)_3.Na(na)_3 \times (nx)_4(na)_4$	1	74	74	75	74	72	3	3	71	4·03
recessive × Edgecote Purple	$(nx)_4(na)_4 \times Nx(nx)_3.Na(na)_3.$	1	31	53	37	47	29	2	8	45	11·91
Liddesdale Lads (self)	$Nx(nx)_3Na(na)_3$ Self	1	65	21	59	27	56	9	3	18	—
Kepplestone Kidney (self)	$Nx(nx)_3Na(na)_3$ Self	1	72	29	69	32	63	9	6	23	—
Edgecote Purple (self)	$Nx(nx)_3Na(na)_3$ Self	1	30	18	35	13	29	1	6	12	—
Di Vernon × Liddesdale Lads	$(nx)_4.Na(na)_3 \times Nx(nx)_3.Na(na)_3$	2	46	60	71	35	45	1	26	34	—
Cardinal × Pepo.	$(Nx)_3(nx)_3.Na(na)_3 \times (nx)_4(na)_4$	1	26	6	18	14	17	9	1	5	—
President × Cardinal	$(nx)_4(na)_4 \times (Nx)_3(nx)_3.(Na(na)_3.$	1	40	14	27	27	27	13	0	14	—
Cardinal (self)	$(Nx)_3(nx)_3.Na(na)_3$ Self	1	62	6	52	16	50	12	2	4	—

TABLE 4

Progenies related to U.S.D.A. seedling 41956

Progeny	Parentage	Parental types	Observed			Theoretical Ratio	χ^2	P>	Parental constitution Dominant genes only
			Resistant	Necrotic	Susceptible				
1189	41956 × Katahdin	resistant × susceptible	69	0	85	1 : 0 : 1	1·662	0·1	Rx : recessive
1190	41956 × Shamrock	resistant × susceptible	49	0	55	1 : 0 : 1	0·346	0·5	Rx : recessive
2324	1189(47) Self	resistant	144	0	46	3 : 0 : 1	0·063	0·8	Rx : Rx
—	various × 1189 derivatives	susceptible × resistant	188	0	166	1 : 0 : 1	1·367	0·2	recessive : Rx
—	1189 derivatives × various	resistant × susceptible	324	0	331	1 : 0 : 1	0·075	0·7	Rx : recessive
902	41956 × Kepplestone Kidney	resistant × necrotic	9	7	10	2 : 1 : 1	3·153	0·2	Rx : Nx
1575	902(31) × Flourball	resistant × susceptible	69	43	35	2 : 1 : 1	1·381	0·5	Rx, Nx : recessive
—	1575 derivatives × Flourball	resistant × susceptible	50	37	31	2 : 1 : 1	3·356	0·1	Rx, Nx : recessive

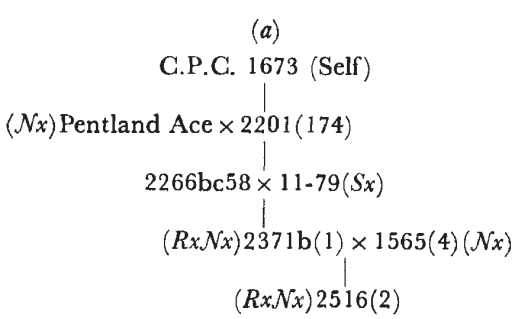
is no indication of linkage in any of three progenies. This result is in contrast to that of Howard and Fuller (1965) who presented evidence that in this variety Nx, Na and Nc are in the same linkage group but in different homologous chromosomes.

(b) *Comprehensive resistance to virus X*

(i) *U.S.D.A. seedling 41956.* (Table 4.) Immunity from virus X was first recorded in the U.S.D.A. seedling 41956 (Schultz and Raleigh, 1933) and there is ample evidence of its comprehensive nature in relation to the many definitive strains of the virus (Cockerham, 1943*b*, 1952; Cockerham and Davidson, 1963; Ross, 1952; Salaman, 1938). The term " immune ", however, became suspect when localised necrotic lesions were observed on the leaves of 41956 and its derivatives in response to infection with the virus (Benson and Hooker, 1960*a*, *b*; Cockerham, 1952; Hutton and Wark, 1952) and the term " extreme resistance " (Ross, 1954*a*, *b*, 1960) is now used frequently to designate this form of resistance.

Inheritance data relating to seedling 41956, table 4, fit the hypothesis that a single gene conditions resistance to virus X in this material. This is in accord with the view of Mills (1965) who provided critical evidence against the earlier interpretation of Stevenson, Schultz and Clark (1939) that the resistance was controlled by two complementary genes. Support for Mills' view was obtained from an examination of 59 crosses between 23 female and seven male parents within the susceptible seedlings of progenies 1189 and 1190 listed in table 4. Fifty-eight of these crosses gave rise to entirely susceptible progeny and in only one case was resistance encountered, namely, in four plants of a progeny of 60 seedlings. This proportion is entirely beyond the limits of 5 resistant: 7 susceptible ratio expected of a cross between AA and B type parents or the 1 resistant: 3 susceptible ratio of a cross between A and B type parents and it is contended, therefore, that the exceptional resistant plants were the produce of illegitimate pollen and not of complementary gene action.

(ii) Solanum tuberosum *subsp.* andigena *C.P.C. 1673.* (Table 5.) Extreme resistance to virus X has frequently been noted in clones of *S. andigena* and particularly in material derived from C.P.C. 1673 in the course of breeding for resistance to potato root eelworm (Dunnett, 1957; Wiersema, 1961). I am indebted to Dr Dunnett for the provision of several progenies derived from C.P.C. 1673 for analysis of reaction to infection with virus X. The essential pedigrees of the key seedlings 2516(2), 2356(12), 2534(43 and 94) and 2371b(1) are given below.



(*b*)

(*Sx*)Kerr's Pink × C.P.C. 1673

|

(*Nx*)Craigs Defiance × 2296(2)

|

(*RxNx*)2356(12)

(*c*)

(*RxNx*)2371b(1) × 2048a(8)(*Nx*)

|

(*RxNxNx*)2534(43 and 94)

In the pedigrees the genetic constitution of the parents with regard to the genes Rx and Nx, is given from information provided from parental reaction to infection with virus X, from external genetical information on the parents, and from the results of the present study given in table 5. The Nx gene in every case is derived from Craigs Defiance either directly or through its derivative variety Pentland Ace.

From the pedigrees it is abundantly clear that the extreme resistance of the progenies examined stems directly from C.P.C. 1673 and, furthermore, from the coherent body of results in table 5, that a single gene, Rx, controls the resistance. Two further conclusions may be drawn. Firstly, from the evidence of progenies 3439, 3987 and 2779 which involve seedlings 2534(94) and 2534(43) of the constitution Rx, Nx, Nx, it is clear that since susceptible seedlings occur the genes Rx and Nx are at different loci and hence are not alleles. Secondly, it will be seen that progeny 2548 is derived from seedling X792/94, an American seedling containing a gene Rx which is derived from U.S.D.A. seedling 41956. The segregations observed for progenies 4715, 4177 and 4178 indicate the presence of two allelic Rx genes, one derived from each parent of 2548, and hence it is logical to conclude that the Rx genes of 41956 and C.P.C. 1673 are identical.

(iii) S. tuberosum *subsp.* andigena *lines C.P.C. 141, 189 and 244.* (Table 6.) In an evaluation of the reactions to viruses of early clones within the Commonwealth Potato Collection (Cockerham, 1943*b*), 28 clones of *S. tuberosum* subsp. *andigena* were found to give null or hypersensitive reactions to both virus X^L and virus X^B. Some years later true seed derived from sib-matings between selfs of three of these clones, C.P.C. 141, C.P.C. 189, C.P.C. 244, was obtained from the curator of the collection for further examination. From C.P.C. 141 five seedlings were raised and these when tested with the two strains of virus X showed four plants to have extreme resistance and one plant to be susceptible to both viruses. This result indicated that a gene of the Rx type was present in the material but the four seedlings carrying the gene were so unthrifty that no further use was made of them. From 10 seeds of C.P.C. 244 five plants were raised and four were found to be extremely resistant to both viruses whilst the fifth gave necrotic, hypersensitive reactions to both viruses. Two of the extremely resistant seedlings, 3638(2) and 3638(5), were used as female parents in crosses with the susceptible variety Flourball. When tested with virus X^L and virus X^B the progeny of seedling 3638(2) segregated into 30 resistant seedlings and six susceptible seedlings, indicative of the duplex constitution $RxRx(rx)_2$.

TABLE 5

Progenies relating to S. andigena *C.P.C. 1673*

Progeny	Parentage	Parental Types	Observed			Theoretical Ratio	χ^2	P>	Parental constitution Dominant genes only
			Resistant	Necrotic	Susceptible				
3975	Majestic × 2516(2)	susceptible × resistant	48	25	15	2 : 1 : 1	2·999	0·2	recessive : Rx, Nx
5115	1591b(9) × 2516(2)	susceptible × resistant	51	12	21	2 : 1 : 1	5·786	0·05	recessive : Rx, Nx
3972	seedling × 2516(2)	resistant × resistant	48	9	8	6 : 1 : 1	0·099	0·95	Rx : Rx, Nx
3969	2356/12 × 2516(2)	resistant × resistant	148	43	13	12 : 3 : 1	0·891	0·5	Rx, Nx : Rx, Nx
3439	2534(94) × Dr McIntosh	resistant × susceptible	20	20	8	6 : 5 : 1	4·667	0·05	Rx, NxNx : recessive
3987	2534(94) × 2516(2)	resistant × resistant	68	24	2	36 : 1 : 1	0·370	0·8	Rx, NxNx : Rx, Nx
2779	2534(43) × 11-79	resistant × susceptible	35	19	6	6 : 5 : 1	2·473	0·2	Rx, NxNx : recessive
2728	2371b(1) × B24/78	resistant × susceptible	19	9	16	2 : 1 : 1	3·045	0·2	Rx, Nx : recessive
2548	2371b(1) × X792/94	resistant × resistant	54	6	4	6 : 1 : 1	3·250	0·1	Rx, Nx : Rx
4222	X792/44 Self	resistant	88	0	34	3 : 0 : 1	0·536	0·3	Rx : Rx
4175	2548(7) × Flourball	resistant × susceptible	30	0	9	5 : 0 : 1	1·123	0·2	RxRx : recessive
4177	2548(31) × Flourball	resistant × susceptible	31	0	6	5 : 0 : 1	0·006	0·9	RxRx : recessive
4178	2548(44) × Flourball	resistant × susceptible	26	0	3	5 : 0 : 1	0·832	0·3	RxRx : recessive
4176	2548(24) × Flourball	resistant × susceptible	25	9	12	2 : 1 : 1	0·913	0·5	Rx, Nx : recessive
4179	2548(46) × Flourball	resistant × susceptible	20	8	9	2 : 1 : 1	0·297	0·8	Rx, Nx : recessive

Seedling 3638(5), on the other hand, produced 21 seedlings resistant, six seedlings hypersensitive and nine seedlings susceptible to both viruses, thus indicating the presence of Rx together with, but independent of, a gene controlling hypersensitive reactions to both strain X^L and strain X^B. This latter gene, hitherto unencountered, was found also in the seedling progeny derived from C.P.C. 189. In this case the progeny produced 26 seedlings necrotic and three seedlings susceptible to both viruses. Twenty-two of the necrotic-reacting seedlings were used as female parents in crosses with susceptible varieties and the progenies raised were tested with virus strains X^L and X^4. Segregations were identical for both viruses and they indicate that a gene with comprehensive effect is concerned and that three of the seedling parents were triplex, eleven were duplex and eight were simplex for this gene.

Observations on the three triplex individuals are recorded in table 6, progenies 4035, 4049 and 4052. In the first two of these progenies a small number of susceptible seedlings was found, exceptional to the expectation. A possible interpretation of their occurrence is that double reduction takes place at the locus concerned. If this is the case, then it is clear from the data that the frequency of double reduction is small since in the few cases where exceptionsl susceptible plants occur the numbers recorded are well below the expectations of complete random chromatid pairing.

The progenies 4055, 4167, 4166 in table 6 refer to associations of the same gene with a gene for extreme resistance to virus X which originated in *Solanum acaule*, the parent 44/1016/10 being a seedling of the fifth generation of a cross between a resistant *S. acaule* plant with *S. tuberosum* (Ross *in litt.*). Progeny 2074 indicates the presence of a single gene in 44/1016/10 and progeny 4055 is compatible with the expectation of a cross between this parent and a parent triplex for the necrosis-inducing gene. The progeny shows again an exceptional susceptible individual and also a discrepancy from the 1 resistant: 1 necrotic ratio expected from the known constitutions of the two parents. This type of discrepancy was found in the progenies of many crosses involving both Rx genes and genes inducing necrosis. It is accounted for by the difficulty in differentiating between resistant individuals showing exceptional localised necrosis (see (b) (i)) and hypersensitive individuals showing very little or no necrosis through physiological effects operating at the time of symptom production. Relatively few plants in each progeny give rise to the difficulty but it is acknowledged that misclassification has taken place in this and other progenies of mixed parentage but in no case does it prejudice the interpretation of the observations.

Progeny 4167 gives clear indication of the relationship between the two genes. The combined total of resistant and necrotic seedlings was 111 to 1 susceptible seedling, the latter being exceptional, and it is evident that the parent 4055(31) possessed three alleles of which one controlled extreme resistance and the other two controlled the necrotic reaction. The indication is, in fact, that the resistance gene from *S. acaule* is allelic with and dominant to the gene for hypersensitivity in *S. andigena* and confirmation of this relationship will be given in the next secion. A similar, and probably identical, pair of alleles designated X^1 and X^n have already been described from direct investigation of *S. acaule* (Cockerham, 1958) and these symbols are used temporarily in both text and tables to distinguish them from the Rx gene of *S. andigena* and the Nx gene of *S. tuberosum*.

TABLE 6

Progenies related to C.P.C. 189

Progeny	Parents	Parental types	Observed			Theoretical Ratio	Suggested genotypes of parents
			Resistant	Necrotic	Susceptible		
3637	C.P.C. 189 sib.		—	26	3	0 : 11 : 1	$X^nX^nxx \times X^nxxx$
4035	3637(1) × Dr McIntosh	necrotic × susceptible	—	266	4	0 : ∞ : 0	$X^nX^nX^nx \times xxxx$
4049	3637(23) × Dr McIntosh	necrotic × susceptible	—	146	1	0 : ∞ : 0	$X^nX^nX^nx \times xxxx$
4052	3637(27) × Dr McIntosh	necrotic × susceptible	—	67	0	0 : ∞ : 0	$X^nX^nX^nx \times xxxx$
2074	1591b(9) × 44/1016/10	susceptible × resistant	53	0	48	1 : 0 : 1	$xxxx \times X^ixxx$
4055	3637(1) × 44/1016/10	necrotic × resistant	30	19	1	1 : 1 : 0	$X^nX^nX^nx \times X^ixxx$
4167	4055(31) × 2986(1)	resistant × susceptible	50	61	1	1 : 1 : 0	$X^iX^nX^nx \times xxxx$
4166	4055(25) × Flourball	resistant × susceptible	184	50	0	5 : 1 : 0	$X^iX^iX^nX^n \times xxxx$

Progeny 4166 is difficult to interpret. The absence of susceptible seedlings in the progeny indicates that the female parent was at least triplex at the X^1 locus but the distribution of 184 resistant to 50 necrotic seedlings does not conform with a parental constitution of $X^1X^nX^nx$ unless there was considerable misclassification of the progeny. An alternative explanation, offered with reserve, is that seedling 4055(25) had a double X^1 gene due to double reduction at the locus concerned in its own parent 44/1016/10. The data fit this interpretation but there is no supporting evidence other than that of the exceptional occurrence of susceptible seedlings which are indicative of double reduction in this and related material.

(iv) Solanum tuberosum *subsp.* andigena *var. Collajera.* (Table 7.) The *andigena* variety Collajera was received from F. Brann, Israel, as a potential parent in breeding for resistance to leaf roll. In the course of commercial breeding it was found that towards virus X both resistant and necrotic seedlings occurred in progenies of which the other parent was susceptible to this virus. The variety was examined in further detail, therefore, and the results are given in table 7.

The observed reactions of seedlings of Collajera selfed (progeny 3818) and crossed with seedlings of known constitution (progenies 4015, 4022, 4024, 3098, 3093 and 3576) provide data which are consistent with the view that the variety possesses a gene for extreme resistance to virus X inherited independently of a gene, present in the duplex condition, controlling necrosis to the virus. A further study was made by crossing with susceptible Dr McIntosh eight putatively resistant seedlings within progeny 3093 derived from Collajera × 44/1016/10, the latter carrying the gene X^1. The results show that one progeny, 3786, was a misclassified necrotic reactor duplex for the necrosis inducing gene whilst the remaining seven progenies disclosed four different constitutions of resistant parents. Thus progeny 3787 indicated two genes for resistance inherited independently, that is, Rx from Collajera and X^1 from 44/1016/10. Progenies 3789, 3791 and 3795 were all similar and indicative of a pair of alleles, X^1 and X^n, thus confirming the results already discussed for progenies 4167 and 4168. Progenies 3785 and 3788 each showed greater proportions of resistant seedlings than the above and their segregations accord well with the view that the genic content of their female parent is X^1X^n with an additional and independent Rx. The remaining progeny, 3794, produced no susceptible seedlings and this, together with the ratio of resistant/necrotic seedlings is consistent with interpretation of the constitution of its female parent as $X^1X^nX^n$ with an independent Rx. Thus, within seven progenies, there was revealed four of the eight resistant genotypes expected from a cross between $Rx(rx)_3 : X^nX^n(x)_2$ with $X^1(x)_3$.

(v) *Relationships between the genes Rx, X^i, X^n, Nx and Nb.* (Tables 8 and 9.) From the evidence of sections (b) (i) and (b) (ii) it is clear that the Rx gene common to U.S.D.A. seedling 41956 and *andigena* clone C.P.C. 1673 is inherited independently of the gene Nx of *S. tuberosum.* It is also clear that Rx genes occur in the andigena clones C.P.C. 141, C.P.C. 244 and Collajera and that in the latter there is also a gene X^n which is inherited independently of Rx (sections (b) (iii) and (b) (iv)). Furthermore, from tables 6 and 7, there is clear evidence that X^n is allelic with the gene X^1 derived from *S. acaule.*

Direct information on the relationship between X^1 and Nx was obtained

TABLE 7

Progenies related to Collajera

Progeny	Parents	Parental types	Observed			Theoretical Ratio	Parental constitution Dominant genes only
			Resistant	Necrotic	Susceptible		
3818	Collajera (Self)	resistant	131	42	3	108 : 35 : 1	Rx, X^nX^n
4015	3637(3) × Collajera	necrotic × resistant	15	14	1	12 : 11 : 1	X^n × Rx, X^nX^n
4022	3638(2) × Collajera	resistant × resistant	64	7	0	66 : 5 : 1	RxRx × Rx, X^nX^n
4024	3638(5) × Collajera		51	36	2	36 : 11 : 1	Rx, X^n × Rx, X^nX^n
3098	Collajera × X792/4		138	13	6	18 : 5 : 1	Rx, X^nX^n × Rx
3093	Collajera × 44/1016/10		208	45	6	18 : 5 : 1	Rx, X^nX^n × X^i
3576	Collajera × 2074(2)		230	35	20	18 : 5 : 1	Rx, X^nX^n × X^i
3786	3093(15) × Dr McIntosh	necrotic × susceptible	0	71	23	0 : 5 : 1	X^nX^n × recessive
3787	3093(21) × Dr McIntosh	resistant × susceptible	66	0	23	3 : 0 : 1	Rx, X^i × recessive
3789	3093(30) × Dr McIntosh		47	37	13	3 : 2 : 1	X^iX^n × recessive
3791	3093(45) × Dr McIntosh		46	28	18	3 : 2 : 1	X^iX^n × recessive
3795	3093(125) × Dr McIntosh		51	32	14	3 : 2 : 1	X^iX^n × recessive
3785	3093(14) × Dr McIntosh		75	13	5	9 : 2 : 1	X^iX^n, Rx × recessive
3788	3093(22) × Dr McIntosh		66	15	5	9 : 2 : 1	X^iX^n, Rx × recessive
3794	3093(121) × Dr McIntosh		69	22	0	9 : 3 : 0	$X^iX^nX^n$, Rx × recessive

from a cross between International Kidney (Nx) and seedling 44/1016/10 (X^1). The resultant progeny 3092, when screened by spraying with virus X^L, yielded 73 resistant and 66 necrotic or susceptible plants, no distinction being recorded between the latter two reactions. From the resistant plants, 11 selections were crossed with Dr McIntosh which is recessive for both genes. Upon test with virus X^L, eight of the progenies obtained segregated into resistant and susceptible types only (103 resistant: 105 susceptible) but three progenies (3775, 3783 and 3784) showed resistant, necrotic and susceptible seedlings (table 8). In each case, there is a good fit to the 2 resistant: 1 necrotic: 1 susceptible ratio indicative of independent inheritance of the two genes concerned.

For information on the relationship between the genes X^n and Nb use was made of the seedling 3637(1), triplex for the gene X^n, in reciprocal crosses with parents carrying the gene Nb. Seedlings of the F_1 progenies were crossed with the susceptible variety Flourball and pilot progenies raised from the seed thus obtained were tested with virus strains X^B and X^L to detect possible combinations of double X^n with single Nb. From such a combination it is possible to differentiate between allelomorphy and independence of the two genes since if allelic, and in the absence of exceptional behaviour, the phenotypic expectation is 5 seedlings necrotic to X^B and X^L to 1 seedling necrotic to X^B only whereas, if the two genes are independent the expected segregation is 10 seedlings necrotic to both strains of virus, 1 seedling necrotic to X^B only, 1 seedling necrotic to X^L only. Of 19 progenies tested, nine showed the desired combination of the two genes and three progenies, 5379, 5393 and 5603, were tested extensively with both viruses. The results, table 9, show that each progeny fitted closely to the 10 : 1 : 1 ratio indicative of independent loci.

(vi) *Di-haploid material.* (Tables 10, 11 and 12.) The early work of Hougas and Peloquin (1957, 1958*a*, 1958*b*, 1960) directed attention to the potential use of di-haploids of tetraploid potatoes as material suitable for the simplification of genetic studies. By using *S. phureja* and other diploid species as pollen parents many di-haploids have been produced including plants containing the genes Rx, derived from U.S.D.A. seedling 41956, Rx from C.P.C. 1673, Nb from Katahdin and Nx from Cardinal. Unfortunately, no success has been obtained in intercrossing between these di-haploids and the only information yet available from this class of material is the product of two crosses between (1) a di-haploid carrying the gene Nx from Cardinal and a diploid seedling carrying a gene Nx from *S. sparsipilum*, C.P.C. 71, and (2) between a di-haploid carrying the gene Rx derived from C.P.C. 1673 and the same seedling derived from *S. sparsipilum*.

The variety Cardinal is duplex for the gene Nx (Cadman, 1942; Cockerham, 1943*a*) and from pollination with *S. phureja*, C.P.C. 979, six di-haploids were obtained of which five are either homozygous or heterozygous for Nx. A heterozygote, seedling 3837(16), was successfully crossed with a diploid seedling 1764(15) also heterozygous for a gene Nx derived from *S. sparsipilum*, C.P.C. 71, and which conditions necrotic reactions to X^L with susceptibility to X^B. In this respect the gene is similar to the Nx of *S. tuberosum* in general and the variety Cardinal in particular. As is seen from table 10 the result of testing the progeny of the original cross and of some of its constituent seedlings in crosses with recessive seedlings is to indicate that the genes from Cardinal and *S. sparsipilum* are alleles and probably identical.

TABLE 8

Relationship between X^i and Nx

Progeny	Parental	Parental types	Observed: Resistant	Observed: Necrotic	Observed: Susceptible	Theoretical Ratio	Parental constitution Dominant genes only
3092	International Kidney × 44/1016/10	necrotic × resistant	73	66		2 : 1 : 1	(Nx) × X^i
various	3092 selections × Dr McIntosh	resistant × susceptible	103	0	108	1 : 0 : 1	X^i × recessive
3775	3092(27) × Dr McIntosh	resistant × susceptible	33	19	18	2 : 1 : 1	X^i, Nx × recessive
3783	3092(51) × Dr McIntosh	resistant × susceptible	37	14	17	2 : 1 : 1	X^i, Nx × recessive
3784	3092(68) × Dr McIntosh	resistant × susceptible	12	5	7	2 : 1 : 1	X^i, Nx × recessive

TABLE 9

Relationship between X^n and Nb

Progeny	Parentage	Parental types	Observed Reactions to X^B/X^L: nec/nec	nec/sus	sus/sus	Theoretical ratio	Parental constitution Dominant genes only
4384	2070(9) × 3637(1)	necrotic × necrotic	20	0	0	∞ : 0 : 0	Nb × $X^nX^nX^n$
5379	4384(6) × Flourball	necrotic × susceptible	153	14	19	10 : 1 : 1	X^nX^n, Nb × recessive
5393	4384(23) × Flourball	necrotic × susceptible	143	14	15	10 : 1 : 1	X^nX^n, Nb × recessive
4035	3637(1) × Dr McIntosh	necrotic × necrotic	266	0	4	∞ : 0 : 0	$X^nX^nX^n$ × Nb
5603	4035(12) × Flourball	necrotic × susceptible	63	7	6	10 : 1 : 1	X^nX^nNb × recessive

TABLE 10

Progenies derived from di-haploid of Cardinal and a derivative of S. sparsipilum

Progeny	Parentage	Parental type	Observed: Necrotic	Observed: Susceptible	Theoretical ratio	Constitution of parents Dominant genes only
4204	US-W 88 × 1764(15)	susceptible × necrotic	43	43	1 : 1	recessive × Nx
3852	1764(15) × US-W 1268	necrotic × susceptible	32	30	1 : 1	Nx × recessive
5034	3837(16) × 1764(15)	necrotic × necrotic	29	11	3 : 1	Nx × Nx
5091	5034(2) × 3299(1)	susceptible × susceptible	0	36	0 : ∞	recessive × recessive
5096	(16) × 3299(1)	susceptible × susceptible	0	16	0 : ∞	recessive × recessive
5094	(13) × 3299(1)	necrotic × susceptible	11	14	1 : 1	Nx × recessive
5095	(15) × 3299(1)		8	20	1 : 1	Nx × recessive
5099	(37) × 3299(1)		7	4	1 : 1	Nx × recessive
5100	(39) × 3299(1)		13	14	1 : 1	Nx × recessive
5101	(40) × 3299(1)		16	15	1 : 1	Nx × recessive
5103	(36) × 3299(1)		6	5	1 : 1	Nx × recessive
5092	(7) × 3299(1)		45	0	∞ : 0	NxNx × recessive
5098	(33) × 3299(1)		72	0	∞ : 0	NxNx × recessive

TABLE 11

Linkage in di-haploid material derived from Cardinal

Ref. No.	Parents	Genetic type	Single factor segregation: Virus X nec	Virus X sus	Virus A nec	Virus A sus	Joint segregation X/A reactions: nec/nec	nec/sus	sus/nec	sus/sus	Recombination %
5034	3837(16) × 1764(15)	Nxnx.Nana. × Nxnx.nana	22	10	18	14	16	6	2	8	—
5583	5034(1) × 3309(1)	Nxnx.Nana. × nxnx.nana	14	8	13	9	11	3	2	6	—
5587	5034(11) × 3309(1)	Nxnx.Nana. × nxnx.nana	9	7	8	8	7	2	1	6	—
5094	5034(13) × 3299(1)	Nxnx.Nana. × nxnx.nana	10	17	12	15	9	1	3	14	—
5095	5034(15) × 3299(1)	Nxnx.Nana. × nxnx.nana	26	24	26	24	23	3	3	21	12·00
Total	4 progenies	Nxnx.Nana. × nxnx.nana	59	58	59	58	50	9	9	47	15·65

TABLE 12

Relationship between Rx and Nx in di-haploid material

Progeny	Parentage	Parental type	Observed: Resistant	Necrotic	Susceptible	Theoretical ratio	Parental constitution Dominant genes only
4424	4163 × C.P.C. 51a	resistant × susceptible	14	0	15	1 : 0 : 1	Rx × recessive
4422	4163 × 1764(15)	resistant × necrotic	25	26	14	2 : 1 : 1	Rx × Nx
5071	4422(15) × 3299(11)	resistant × susceptible	13	0	16	1 : 0 : 1	Rx × recessive
5074	(42) × 3299(11)		13	0	15	1 : 0 : 1	Rx × recessive
5078	(74) × 3299(11)		7	0	19	1 : 0 : 1	Rx × recessive
5073	(29) × 3299(11)		12	7	6	2 : 1 : 1	Rx, Nx × recessive
5075	(44) × 3299(11)		16	6	6	2 : 1 : 1	Rx, Nx × recessive
5076	(64) × 3299(11)		14	8	8	2 : 1 : 1	Rx, Nx × recessive
5079	(116) × 3299(11)		4	4	2	2 : 1 : 1	Rx, Nx × recessive
5080	(128) × 3299(11)		16	8	6	2 : 1 : 1	Rx, Nx × recessive

An examination of linkage between the Nx and Na loci in di-haploid material was made in four small progenies derived from progeny 5034, the relevant data being given in table 11. The data indicate clearly that the two loci are linked in the coupling phase with a mean recombination value of 15·6 per cent., a value which is very much greater than that of 5·43 per cent. recorded for the tetraploid material.

Seedling 4163 was the only haploid derived from a pollination of seedling 2543(94) (see progeny (*c*) in (b) (ii)) with *S. multidissectum*. On test the seedling was found to be comprehensively resistant to virus X and hence to be in possession of the Rx gene of C.P.C. 1673. The data from progeny 4424 (table 12) indicate that it is heterozygous for this gene. The further data of table 12 were obtained from a cross between 4163 and seedling 1764 (15) which carries the Nx gene of *S. sparsipilum* and they indicate clearly (progenies 5073, 5075, 5076, 5079 and 5080) that the Rx and Nx genes are inherited independently. Since the Nx gene concerned is allelic with the Nx of *S. tuberosum* this result confirms the conclusion drawn from the examination of tetraploid data in section (b) (ii).

(c) S. chacoense *and* S. microdontum. (Table 13.)

(i) *Relationship of genes controlling necrotic hypersensitive reactions to virus Y.* The diploid clones *S. chacoense* (C.P.C. 51B) and *S. microdontum* (C.P.C. 51A) are both sources of comprehensive resistance to virus Y which have been used as basic material in commercial potato breeding. In each case the fundamental reaction to infection with virus A, virus C and representative strains of virus Y is one of necrotic hypersensitivity which in intensity may vary from almost imperceptible localised lesions to full systemic lethal necrosis according to the varied circumstances of the clone concerned and the manner of inoculation.

A detailed study of the character in C.P.C. 51A was made by Ganguly (1949) who concluded that single genes control necrotic reactions to viruses A, C and Y and that the three genes concerned are closely linked to each other and also to the gene Nx of *S. sparsipilum*. Inheritance data from a limited study of the reactions of C.P.C. 51B to infection with a common strain of virus Y are given in section (*a*) of table 13. The segregation of necrotic : susceptible seedlings in each progeny is typical of that for a single gene controlling the necrotic reaction and present in the heterozygous condition in each necrosis-reacting parent.

The relationship between the Ny genes of C.P.C. 51A and C.P.C. 51B is examined in section (*b*) of table 13. Progeny 3853 is relevant to the other progenies only in that it related to seedling 614(7) which is derived from C.P.C. 51A × C.P.C. 1311, the latter being a clone of *S. phureja* susceptible to viruses A, C and Y. Each plant in this progeny was tested with viruses A, C and Y and the complete identity of the reactions to all three viruses gives confirmation of the comprehensive nature of the single gene concerned. Seedling 1272(26) is also an uncomplicated derivative of C.P.C. 51A and it is evident from progeny 2984 that in the mating of this seedling with C.P.C. 51B two allelic genes were brought together in the clone 1716(2). The direct cross of C.P.C. 51A and C.P.C. 51B, progenies 4420 and 5066, confirmed this allelism and the probable identity of the genes from each source.

This relationship suggested to Professor J. G. Hawkes that the character

TABLE 13

Progenies related to S. chacoense *C.P.C. 51B and* S. microdontum *C.P.C. 51A*

	Progeny	Parentage	Parental reactions	Necrotic	Susceptible	Theoretical ratio	Parental constitution Dominant genes only
(*a*)	2514	C.P.C. 51B × C.P.C. 1311	necrotic × susceptible	16	14	1 : 1	Ny × recessive
	2513	C.P.C. 1311 × C.P.C. 51B	susceptible × necrotic	10	10	1 : 1	recessive × Ny
	4419	H18(3) × C.P.C. 51B	susceptible × necrotic	19	17	1 : 1	recessive × Ny
	2610	2514(7) × seedling	necrotic × susceptible	15	20	1 : 1	Ny × recessive
	2594	C.P.C. 51B × 2514(4)	necrotic × necrotic	12	4	3 : 1	Ny × Ny
	—	seedlings × 2513(3)	necrotic × necrotic	44	14	3 : 1	Ny × Ny
	—	seedlings × 2514(1)	necrotic × necrotic	129	39	3 : 1	Ny × Ny
	—	seedlings × 2514(6)	necrotic × necrotic	73	24	3 : 1	Ny × Ny
(*b*)	3853	US-W88 × 614(7)	susceptible × necrotic	21	24	1 : 1	recessive × Ny
	1716	1272(26) × C.P.C. 51B	necrotic × necrotic	2	0	3 : 1	Ny × Ny
	2984	1716(2) × C.P.C. 1311	necrotic × susceptible	32	0	∞ : 0	NyNy × recessive
	4420	C.P.C. 51A × C.P.C. 51B	necrotic × necrotic	9	2	3 : 1	Ny × Ny
	5066	4420(1) × 1764(4)	necrotic × susceptible	95	0	∞ : 0	NyNy × recessive

of hypersensitivity to virus Y might be used to study introgression between the two species, *S. chacoense* and *S. microdontum*, which, although dissimilar in taxonomic character, have overlapping but not identical ranges of distribution. To this end, 48 samples of *S. chacoense* and 27 samples of *S. microdontum* have been examined and hypersensitive response to virus Y found in 32 samples of *S. chacoense* and 11 samples of *S. microdontum*. As yet, however, no significant observations on introgression can be recorded.

(ii) *Linkage studies.* (Tables 14 and 15.) In his study on the inheritance of reactions to viruses, in particular diploid species of potato, Ganguly (1949) recorded linkage between a gene Ny controlling hypersensitive reaction to virus Y in *S. microdontum*, C.P.C. 51A, and a gene Nx controlling hypersensitive reaction to virus X in *S. sparsipilum*, C.P.C. 71. This observation is confirmed in two progenies, 4999 and 5000 (section (*a*) table 14) in which the Ny gene from C.P.C. 51A was received *via* seedling 614(7) and the Nx gene from C.P.C. 71 *via* seedling 1764(15). It is again confirmed in section (*b*) of table 13 where data relating to 2984(30) × C.P.C. 71 are given. In this case the Ny gene in 2984(30) may be derived from either C.P.C. 51A or C.P.C. 51B (see previous section (c) (i)).

Apart from the plain evidence of linkage between the *loci* of Ny and Nx in the relevant six progenies of table 14 (*a*) and (*b*), there is a degree of consistency between them in that the recombinants within each amount to approximately 13 per cent. of the seedlings tested.

The progenies comprising table 14 (*c*) relate to three seedlings, H18(1), H18(2) and H18(3) which were derived from the seed sample HPR 56 of *S. chacoense*. Two of these seedlings, H18(1), H18(3), were found to give necrotic, hypersensitive reactions when infected with virus X and to be susceptible to virus Y whereas the third seedling H18(2) was susceptible to virus X and hypersensitive to virus Y. Similar hypersensitive reactions to virus X were found later in 11 of 43 samples of *S. chacoense* and 3 of 27 samples of *S. microdontum* examined in a survey of the two species. The two X-hypersensitive seedlings (H18)1 and H18(3) were each mated with the Y-hypersensitive seedling H18(3) and the resultant progenies, 3180 and 3183, tested by inoculation with virus X. In each case segregations indicative of parents heterozygous for a gene Nx were obtained. A portion only of the seedlings giving necrotic reactions to virus X were tested with virus Y and all were found to be hypersensitive to this virus also. It seems possible, therefore, that H18(2) is homozygous for a gene Ny controlling Y-hypersensitivity. Subsequent progenies were derived by mating three individuals each hypersensitive to both virus X and virus Y with *S. phureja*, C.P.C. 1311, susceptible to both viruses. The seedlings within each progeny were cloned and tested with the two viruses independently. Again, linkage in the repulsion phase was detected but with only 5 per cent. recombinants as compared with the value of 13 per cent. derived from the earlier data. Progenies of a second backcross to a recessive seedling provided information on the coupling phase of linkage which again indicated a low number, 4·74 per cent., of recombinants.

A further investigation into linkage between the Nx gene of *S. chacoense*, H18(3) and the Ny gene of C.P.C. 51B, was made in an examination of progenies 5005, 5015 and 5064, table 14 (*d*). The combined segregations of these progenies again indicate linkage, in the repulsion phase, between

TABLE 14

Linkage between Ny (chacoense *and* microdontum) *and Nx* (sparsipilum), (*a*) (*b*), *and Nx* (chacoense), (*c*) *and* (*d*)

	Progeny	Parentage	Parental constitution	Observations: Single gene segregations — Virus X: nec	Virus X: sus	Virus Y: nec	Virus Y: sus	Combined segregations X/Y reactions: nec/nec	nec/sus	sus/nec	sus/sus	Remarks
(*a*)	3856	1764(15) × 614(7)	Nxnxnyny × nxnxNyny	15	10	9	6	9	6	10		1 : 1 : 2
	4999	3856(24) × 1764(14)	NxnxNyny × nxnxnyny	18	18	18	18	3	15	15	3	Repulsion
	5000	3856(30) × 3309(7)	NxnxNyny × nxnxnyny	10	14	14	10	1	9	13	1	Repulsion
			Recombination Repulsion 13·33%									
(*b*)	3187	2984(30) × C.P.C. 71	nxnxNyny × Nxnxnyny	29	23	25	27	14	15	11	12	1 : 1 : 1 : 1
	3318	3187(6) × C.P.C. 1311	NxnxNyny × recessive	35	31	32	34	6	29	26	5	Repulsion
	3316	3187(4) × C.P.C. 1311	NxnxNyny × recessive	70	52	56	66	9	61	47	5	Repulsion
	5050	3316(23) × 3299(11)	NxnxNyny × recessive	29	29	29	25	24	3	5	22	Coupling
	5061	3316(68) × 3309(7)	NxnxNyny × recessive	42	44	46	40	37	5	9	35	Coupling
			Recombination Coupling 15·71%					Repulsion 13·30%				
(*c*)	3180	H18(1) × H18(2)	Nxnxnyny × nxnxNyNy	4	3	—	—	4	—	—	—	Selections only
	3183	H18(1) × H18(2)	Nxnxnyny × nxnxNyNy	29	30	—	—	10	—	—	—	Selections only
	3291	3180(1) × C.P.C. 1311	NxnxNyny × recessive	21	23	25	19	2	19	23	0	Repulsion
	3292	3180(2) × C.P.C. 1311	NxnxNyny × recessive	49	51	50	50	3	46	47	4	Repulsion
	3301	3138(8) × C.P.C. 1311	NxnxNyny × recessive	65	53	55	63	3	62	52	1	Repulsion
	5047	3292(42) × 3299(14)	NxnxNyny × recessive	49	45	55	30	48	0	6	30	Coupling
	5056	3292(8) × 3309(7)	NxnxNyny × recessive	58	40	56	38	53	5	3	33	Coupling
	5058	3301(94) × 3309(7)	NxnxNyny × recessive	49	47	46	41	40	3	3	38	Coupling
			Recombination Coupling 7·35%					Repulsion 4·96%				
(*d*)	4418	C.P.C. 51B × H18.3	nxnxNyny × Nxnxnyny	—	—	—	—	12	—	—	—	Selections only
	4419	418·3 × C.P.C. 51B	Nxnxnyny × nxnxNyny	20	16	19	17	9	11	10	6	1 : 1 : 1 : 1
	5005	4418(58) × 3309(7)	NxnxNyny × nxnxnyny	66	79	73	72	7	59	66	13	Repulsion
	5015	4419(31) × 3309(7)	NxnxNyny × nxnxnyny	35	43	42	36	0	35	42	1	Repulsion
	5064	4419(6) × 1716(4)	NxnxNyny × nxnxnyny	35	27	27	33	4	31	23	2	Repulsion
			Recombination Repulsion 9·54%									

the two genes but the recombination values were inconsistent at 13·8 per cent., 1·2 per cent. and 7·1 per cent. for the three progenies.

In spite of this latter inconsistency, the different recombination values for material involving Nx from *S. sparsipilum* and Nx from *S. chacoense* cannot be entirely ignored. The genes themselves are undoubtedly different in effect since Nx from *S. sparsipilum* is specific and is brought into action by Group 1 and Group 3 strains of the virus only. Towards Group 2 and Group 4 strains material containing this gene is susceptible in reaction. Nx from *S. chacoense*, on the other hand, is comprehensive in effect and controls hypersensitive reaction to strains within all four groups of virus X. This linkage of both genes with the Ny locus of *S. chacoense* indicates, however, that they are situated on the same chromosome. Progenies derived from intercrosses between seedlings carrying Nx from *S. chacoense* (H18(1) and H18(3)) and those carrying Nx from *S. sparsipilum* provide the data of table 15.

From the evidence of segregation in all the progenies listed in table 15 it is immediately apparent that the two Nx genes are not alleles. Where they are associated, however (progenies 4390, 4392, 4404, 4408, 4394 and 4213), there is a marked deviation from the 3 necrotic : 1 susceptible ratio indicative of independent inheritance. The deviation in each case is towards an excess of necrosis-reacting seedlings and this is in complete conformity with an expectation of 2-*p* necrotic types to *p* susceptible recombinants in a repulsion linkage between the two genes. This again conforms with the evidence of table 14 that both genes are linked with the Ny locus though they are themselves situated at different *loci*.

(d) *Reaction to virus Y in* S. hougasii *and* S. demissum. (Table 16)

In the course of examining wild potato material for reactions to viruses a number of plants of *S. hougasii* = *S. spectabile* aroused interest by their reaction or lack of reaction to virus Y. When response to infection was observed it took the form of necrosis localised in the leaves, in the stems, or at the stem apices of affected plants. In no case was an affected plant killed nor was necrosis observed in any tuber progeny. These null and localised reactions contrast greatly with the rapid and systemic lethal reactions of hypersensitive plants of the related species *S. demissum* in response to infection with virus Y. Attempts were made, therefore, to examine the inheritance of the two forms of reaction in the common background of hybrids between the two species and the appropriate segregations are shown in table 16.

The original material of *S. hougasii* was received in four seed lines, two (progenies 3061 and 3062) obtained from the Inter-Regional Potato Collection, Sturgeon Bay, Wisconsin, U.S.A., and two (progenies 3063 and 3064) from the Institute for Plant Breeding, Gross Lüsewitz, East Germany. When inoculated by graft with virus Y three of these lines segregated in a manner indicative of inter-crosses of parents heterozygous for a single gene, Ry. The fourth line, 3064, indicates a cross between a resistant heterozygote and a susceptible recessive. Two resistant F_1 seedlings crossed with susceptible *S. demissum*, each gave progenies (3262 and 3263) which segregated into equal numbers of resistant and susceptible seedlings. In crosses with necrosis-reacting *S. demissum*, C.P.C. 2103, which is homozygous in one genome for the gene Ny (Cockerham, 1958), equal proportions of resistant and necrotic seedlings were obtained in two progenies (3269 and 3272)

TABLE 15

Progenies relating to Nx (chacoense) *and Nx* (sparsipilum)

Progeny	Parentage	Parental reactions	Observations: Necrotic	Observations: Susceptible	Theoretical ratio	Remarks
3875	US-W149 × 1764(3)	susceptible × necrotic	18	32	1 : 1	
3180	H18(1) × H18(2)	necrotic × susceptible	4	3	1 : 1	
3859	H18(1) × 1764(3)	necrotic × necrotic	11	3	3 : 1	
4399	3859(3) × 1764(4)		30	29	1 : 1	
4401	(10) × (4)		31	24	1 : 1	
4391	(12) × (8)		11	11	1 : 1	
4402	(13) × (8)		25	24	1 : 1	
4406	(13) × (14)	necrotic × susceptible	72	77	1 : 1	
4403	(20) × (4)		30	36	1 : 1	
4407	(20) × (14)		66	57	1 : 1	
4390	(11) × (8)		29	6	3 : 1	necrotics
4392	(15) × (8)		67	4	3 : 1	in excess
3183	H18(3) × H18(2)	necrotic × susceptible	29	30	1 : 1	
3860	H18(3) × 1764(3)	necrotic × necrotic	47	17	3 : 1	
4393	3860(3) × (8)		21	21	1 : 1	
4396	(7) × (8)		45	41	1 : 1	
4217	(8) × 3318(44)		35	39	1 : 1	
4218	(11) × (44)		66	60	1 : 1	
4398	(12) × 1764(8)		73	63	1 : 1	
4404	(1) × (4)	necrotic × susceptible	42	5	3 : 1	
4408	(1) × (8)		39	1	3 : 1	necrotics
4394	(4) × (8)		114	6	3 : 1	in excess
4213	(5) × 3318(44)		15	3	3 : 1	
4413	4213(62) × 1764(8)		38	38	1 : 1	

TABLE 16

Progenies relating to S. hougasii

Progeny	Parentage	Parental reactions	Observed			Parental constitution Dominant genes only
			Resistant	Necrotic	Susceptible	
3061	P.I. 161726 × P.I. 161740	—	13	0	8	Ry × Ry
3262	3061(4) × C.P.C. 1·3	resistant × susceptible	6	0	6	Ry × recessive
3580	3262(4) × 2741	resistant × susceptible	8	0	10	Ry × recessive
3062	P.I. 161740 × P.I. 161726	—	28	0	11	Ry × Ry
3263	3062(35) × C.P.C. 1·3	resistant × susceptible	7	0	7	Ry × recessive
3587	3263(14) × 2741	resistant × susceptible	11	0	12	Ry × recessive
3269	3062(35) × C.P.C. 2103	resistant × necrotic	2	5	0	Ry × NyNy
3902	3269(2) × 2741	resistant × susceptible	6	2	4	Ry, Ny × recessive
4193	3269(2) × 2539(1)	resistant × susceptible	33	9	16	Ry, Ny × recessive
3270	3062(37) × C.P.C. 2103	resistant × necrotic	22	0	0	RyRy × NyNy
3593	3270(4) × 2741	resistant × susceptible	8	3	3	Ry, Ny × recessive
3063	66/3*	—	8	0	1	Ry × Ry
3272	3063(6) × C.P.C. 2103	resistant × necrotic	7	11	0	Ry × NyNy
3907	3272(1) × 2741	resistant × susceptible	10	10	10	Ry, Ny × recessive
4194	3272(1) × 2539(1)	resistant × susceptible	10	7	9	Ry, Ny × recessive
3620	3272(3) × C.P.C. 1·3	resistant × susceptible	10	10	10	Ry, Ny × recessive
4195	3272(3) × 2539(1)	resistant × susceptible	12	11	9	Ry, Ny × recessive
3911	3272(8) × 2741	resistant × susceptible	13	5	5	Ry, Ny × recessive
4196	3272(8) × 2539(1)	resistant × susceptible	19	12	12	Ry, Ny × recessive
3064	66/4*	—	14	0	15	Ry × recessive
3273	3064(29) × C.P.C. 2103	susceptible × necrotic	0	15	0	recessive × NyNy

* Seed sample numbers.

whereas the whole of a third progeny (3270) was resistant to infection. Nine F_2 progenies were raised from matings between resistant F_1 seedlings of the constitution Ry, Ny and susceptible *S. demissum* pollen parents. In every case these progenies segregated into three distinct classes with regard to reaction to virus Y, namely, resistant, necrotic and susceptible, and, moreover, in such proportions, paying due regard to the small numbers concerned, as to be consistent with the view that two independent genes, Ry and Ny, control the resistant and the necrotic reactions.

From replicated tests with viruses A, C and strains of virus Y, upon seedlings of whole progenies or selected from some progenies, there was clear indication that the gene Ry is a single unit with comprehensive effect in relation to all strains of the virus Y complex. In this respect it is similar to the Ny gene of *S. chacoense* and *S. microdontum* (see section (c) and the Ny gene of *S. demissum*. The reactions controlled by Ry, however, differ from those controlled by the Ny genes and appear to be more closely similar to those of comprehensively resistant clones of *S. stoloniferum* (section (e)).

(e) S. stoloniferum, *reactions to viruses Y and A*

S. stoloniferum is a very polymorphic Mexican species with locally abundant forms which, in the past, have themselves been given specific rank, *e.g. S. ajuscoense*, *S. antipoviczii*, *S. longipedicalletum* and *S. malinchense* (Hawkes, 1963). It was under these names that the outstanding characteristic of the species namely, extreme resistance or immunity from virus Y was first reported (Cockerham, 1943*a*, 1951; Hawkes, 1945; Ross and Baerecke, 1950) and its comprehensive nature established (Ross, 1952; Easton, Larson and Hougas, 1958). Comprehensive extreme resistance is, however, only one of seven phenotypic responses to have been recognised in the present studies which, for the most part, relate to the combined reactions of *S. stoloniferum* to virus Y on the one hand and to virus A on the other.

The seven phenotypes classified upon the reactions given to both viruses are as follows:

Phenotype 1. No visible reaction to sap transfers of either virus Y or virus A. Graft transfers with each virus produce varying reactions which range, according to circumstances, from no visible response to a variety of localised necrotic flecks in leaves and/or stems accompanied by stunting of axillary shoots and also of the scion. No virus is recoverable either from the grafted plant itself or from its tuber progeny.

Phenotype 2. Reactions to virus Y exactly similar to those of phenotype 1. Sap inoculations with virus A, however, induces local lesions on the inoculated leaves followed, occasionally, by systemic invasion with lethal top-necrosis. Graft inoculation invariably induces the latter symptoms.

Phenotype 3. Reactions to virus Y exactly similar to those of phenotype 1. Sap inoculations with virus A induce local lesions followed usually by systemic infection with a rusty necrosis affecting leaf margins at first but often progressing over the entire leaf and leading to leaf drop. Eventually, however, the production of heavy necrosis ceases and the plant continues to grow, though weakly, and to exhibit symptoms of a necrotic mosaic. Tubers from such plants give rise to similar weak, necrotic mosaic-affected plants. Graft infection usually causes a severe rusty necrosis which, if induced at an early stage, may kill the plant. In this case it is difficult to differentiate the

reaction from that of phenotype 2 and this leads to a certain amount of misclassification. Where tubers are formed prior to death the two phenotypes may be distinguished by the necrotic, lethal effect on phenotype 2 as compared with the non-lethal effect of phenotype 3 which produce weak plants with a necrotic mosaic from which virus A is recoverable.

Phenotype 4. Sap transfers with virus Y cause local lesions and frequently lethal top necrosis. Graft transfers always cause the latter disease. Sap and graft transfers with virus A produce symptoms of non-lethal, rusty necrosis identical with those produced by this virus on phenotype 3.

Phenotype 5. Similar necrotic reactions to virus Y as in phenotype 4. Virus A is accepted from sap or graft inoculations with the production of very mild symptoms in which there may be a slight mosaic pattern but more usually there is only a slight pallor in leaf colour and a slightly wavy margin to the leaves of infected plants as compared with healthy plants. The virus is recoverable.

Phenotype 6. The reaction to virus Y is one of complete susceptibility to either sap or graft inoculations. Symptoms of infection vary from almost imperceptible to fairly strong mosaic patterns accompanied by reduction in size of organs and puckering of leaves with marginal waviness. The virus is recoverable. Towards virus A this phenotype is similar to phenotypes 3 and 4, *i.e.* it exhibits non-lethal rusty necrosis.

Phenotype 7. Plants completely susceptible to both virus Y and virus A as in phenotypes 6 and 5, respectively.

There are thus three reactions to virus Y, resistant (phenotypes 1, 2, 3), lethal necrotic (phenotypes 4, 5) and susceptible (phenotypes 6, 7) and four reactions to virus A, resistant (phenotype 1), lethal necrotic (phenotype 2), non-lethal, rusty necrosis (phenotypes 3, 4, 6), and susceptible (phenotype 7). In no case have we encountered the following combinations of reaction; resistant to Y/susceptible to A; necrotic to Y/resistant to A; necrotic to Y/lethal necrotic to A; susceptible to Y/resistant to A; and susceptible to Y/lethal necrotic to A.

As a temporary measure, adopted for clarity, it is proposed to symbolise the genes concerned in determining phenotype by the letter *R* bearing a numbered superscript to correspond with phenotype, thus, R^1 will indicate the gene determining phenotype 1. In the tabulated results of infections the parental reactions given relate firstly to virus Y with the symbols res (resistant), nec (necrotic) and sus (susceptible) and secondly to virus A with res, nec, rty (rusty) and sus. Since there is little indication to the contrary, the results are interpreted on a basis of disomic inheritance within an allotetraploid. More detailed studies with larger progenies may reveal a different basis of inheritance as suggested by Ross (1958).

Results

(i) *C.P.C. 9, C.P.C. 28·4 and P.I. 160226, phenotype 1.* (Table 17)

The *S. stoloniferum* clones C.P.C. 9 and C.P.C. 28·4 both gave null reactions when tested with several strains of virus Y and virus A (phenotype 1). On selfing C.P.C. 9 the progeny, 1179, segregated into 7 null reactors: 28 resistant with necrotic flecks: 13 susceptible, when graft inoculated with virus Y. Two null reactors were mated with susceptible seedlings and the progeny tested with virus Y. One failed to segregate, the other segregated in

the proportion of 1 resister : 1 susceptible with some resistant seedlings in each progeny showing nectrotic flecks. Of 16 resistant plants which showed necrotic flecks, 3 failed to segregate and 13 gave segregations of 1 resistant : 1 susceptible when tested with virus Y. Again there were both null reactors and necrotic fleck reactors among the resistant plants of each progeny and there was no indication of differential genic effect on the production of these symptoms. It was concluded, therefore, from this and similar evidence derived throughout the course of the investigation that resistance is under the control of a single gene R^1 and that the presence or absence of necrotic flecks is the result of circumstantial causes operating on a basic hypersensitive response of host plant to virus.

It is clear from the foregoing and from table 17 that C.P.C. 9 is heterozygous for a single gene controlling resistance to virus Y. The sample data for C.P.C. 28·4, on the other hand, show that this clone is homozygous for a similar resistance gene and they also indicate (progeny 3926) that there was present additionally an independent gene which controls the non-lethal rusty necrotic reaction to virus A (phenotype 6). Further evidence of this gene, R^6, occurs in subsequent progenies related to C.P.C. 28·4 and a similar and probably identical gene is disclosed in progeny 3925 and other progenies derived from P.I. 160226. The latter itself is homozygous with respect to a gene for resistance to viruses Y and A. At first this gene was thought to be distinct from those of C.P.C. 9 and C.P.C. 28·4 since the resistant seedlings of progenies 2562, 3358 and 3925 all showed more necrosis, especially towards Y, than those of progenies 2630 and 1179. When the combinations between the three sets of parents were examined, however, it was no longer possible to differentiate derivatives of P.I. 160226. Hence, since the lack of segregation in the F_2 families derived from the combinations (progenies 4531, 4518, 3920 and 3921) indicates that the genes from all three sources are at the same *locus*, it is concluded that they are also probably identical.

(ii) *P.I. 161172, phenotype 2.* (Table 18)

Progenies 2472 and 3936, table 18, were derived from *S. stoloniferum* P.I. 161172. In each case the total progeny was composed of seedlings which were resistant to virus Y but reacted with lethal top necrosis to virus A (phenotype 2). An F_2 progeny, 4244, segregated into three phenotypes, 2, 6, and 7, in proportions suggestive of single gene control of phenotype 2 with an independent gene controlling phenotype 6.

Phenotypes 1 and 2 were brought together to give progeny 3978 and segregation was found in the F_2 progenies 5604 and 4250. Again only three phenotypes were found, namely, phenotypes 1, 2 and 6, in proportions which indicate independence of the genes controlling phenotypes 1 and 2 and the accession from each parent of a gene controlling phenotype 6. With R^6 symbolising the latter and R^2 symbolising the gene in control of phenotype 2 the genotypes of the material are given in the final column of the table.

(iii) *C.P.C. 12, C.P.C. 2092, C.P.C. 2093 and C.P.C. 2094, phenotype 3.* (Table 19)

Sources of phenotype 3, resistance to virus Y coupled with non-lethal rusty necrosis to virus A were found in C.P.C. 12, C.P.C. 2092, C.P.C. 2093 and C.P.C. 2094. The results of appropriate matings with susceptible male

TABLE 17

Progenies related to C.P.C. 9, C.P.C. 28.4 and P.I. 160226

Progeny	Parentage	Reaction of parents		Segregation on infection with							Segregation on infection with Y and A Phenotypes with Y/A reactions							Genotypes Dominant genes only
				Y only			A only											
		Y/A	Y/A	res	nec	sus	res	nec	rty	sus	1 res/res	2 res/nec	3 res/rty	4 nec/rty	5 nec/sus	6 sus/rty	7 sus/sus	
1179	C.P.C. 9 self	res/res		35	0	13	—	—	—	—	—	—	—	—	—	—	—	R^1
2025	1179(30) self	res/res		31	0	8	—	—	—	—	—	—	—	—	—	—	—	R^1
2095	1179(15) × 1179(25)	res/res × sus/sus		17	0	13	17	0	0	13	17	0	0	0	0	0	13	R^1 × recessive
3937	2095(86) × 2722(2)	res/res × sus/sus		—	—	—	18	0	0	7	—	—	—	—	—	—	—	R^1 × recessive
2630	C.P.C. 28·4 self	res/res		73	0	0	30	0	0	0	30	0	0	0	0	0	0	R^1R^1, R^6R^6
3359	C.P.C. 28·4 × 2722(6)	res/res × sus/sus		6	0	0	6	0	0	0	6	0	0	0	0	0	0	R^1R^1, R^6R^6 × recessive
3926	3359(1) × 2722(2)	res/res × sus/sus		34	0	30	34	0	14	16	34	0	0	0	0	14	16	R^1, R^6
2652	P.I. 160226 Self	res/res		25	0	0	25	0	0	0	25	0	0	0	0	0	0	R^1R^1, R^6R^6
3358	2652(4) × 2722(16)	res/res × sus/sus		20	0	0	20	0	0	0	20	0	0	0	0	0	0	R^1R^1, R^6R^6 × recessive
3925	3358(19) × 2722(2)	res/res × sus/sus		17	0	15	17	0	6	9	17	0	0	0	0	6	—	R^1, R^6 × recessive
3979	2095(86) × C.P.C. 28·4	res/res × res/res		6	0	0	—	—	—	—	—	—	—	—	—	—	—	R^1 × R^1R^1, R^6R^6
4518	3979(2) × 2473(2)	res/res × sus/sus		50	0	0	32	0	0	0	32	0	0	0	0	0	0	R^1R^1, R^6 × recessive
3987	2095(86) × 2652(26)	res/res × res/res		12	0	0	—	—	—	—	—	—	—	—	—	—	—	R^1 × R^1R^1, R^6R^6
4531	3987(2) × 2473(2)	res/res × sus/sus		32	0	0	32	0	0	0	32	0	0	0	0	0	0	R^1R^1, R^6 × recessive
3354	2652(4) × C.P.C.28·4	res/res × res/res		6	0	0	—	—	—	—	—	—	—	—	—	—	—	R^1R^1, R^6R^6 × R^1R^1, R^6R^6
3920	3354(4) × 2722(2)	res/res × sus/sus		32	0	0	32	0	0	0	32	0	0	0	0	0	0	R^1R^1, R^6R^6 × recessive
3355	2652(16) × C.P.C. 28·4	res/res × res/res		6	0	0	—	—	—	—	—	—	—	—	—	—	—	R^1R^1, R^6R^6 × R^1R^1, R^6R^6
3921	3355(3) × 2722(2)	res/res × sus/sus		26	0	0	26	0	0	0	26	0	0	0	0	0	0	R^1R^1, R^6R^6 × recessive

TABLE 18

Progenies related to P.I. 161172

Progeny	Parentage	Reaction of parents		Segregation on infection with							Segregation on infection with Y and A Phenotypes with Y/A reactions							Genotypes Dominant genes only
				Y only			A only											
		Y/A	Y/A	res	nec	sus	res	nec	rty	sus	1 res/res	2 res/nec	3 res/rty	4 nec/rty	5 nec/sus	6 sus/rty	7 sus/sus	
2472	P.I. 161172 self	res/nec		30	0	0	0	30	0	0	0	30	0	0	0	0	0	R^2R^2, $R^4(R^4)$
3936	2472(27) × 2722(2)	res/nec × sus/sus		32	0	0	0	32	0	0	0	32	0	0	0	0	0	R^2R^2, $R^4(R^4)$ × recessive
4244	3936(1) × 2722(9)	res/nec × sus/sus		23	0	25	0	23	6	19	0	23	0	0	0	6	19	R^2, R^4 × recessive
3978	2472(26) × C.P.C. 28·4	res/nec × res/res		6	0	0	—	—	—	—	—	—	—	—	—	—	—	R^2R^2, R^4R^4 × R^1R^1, R^4R^4
4250	3978(1) × 2722(9)	res/res × sus/sus		24	0	8	14	10	8	0	14	10	0	0	0	8	0	R^2R^2, R^4R^4 × recessive
5604	3978(1) × 2473(2)	res/res × sus/sus		39	0	11	27	12	6		27	12	0	0	0	6	0	R^1, R^2, R^4R^4 × recessive

TABLE 19

Progenies related to C.P.C. 12, C.P.C. 2092, C.P.C. 2093 and C.P.C. 2094

Progeny	Parentage	Reaction of parents Y/A	Reaction of parents Y/A	Segregation on infection with: Y only res	Y only nec	Y only sus	A only res	A only nec	A only rty	A only sus	Segregation on infection with Y and A Phenotypes with Y/A reactions: 1 res/res	2 res/nec	3 res/rty	4 nec/rty	5 nec/sus	6 sus/rty	7 sus/sus	Genotypes Dominant genes only
2471	C.P.C. 12 Self	res/rty		20	0	0	0	0	20	0	0	0	20	0	0	0	0	R^3R^3
3934	2471(16)×2722(2)	res/rty×sus/sus		30	0	0	0	0	30	0	0	0	30	0	0	0	0	$R^3R^3\times$recessive
4242	3934(31)×2722(9)	res/rty×sus/sus		17	0	31	0	0	17	31	0	0	17	0	0	0	31	$R^3\times$recessive
2631	C.P.C. 2092 Self	res/rty		32	0	0	0	0	32	0	0	0	32	0	0	0	16	R^3R^3
3360	C.P.C. 2092×2722(6)	res/rty×sus/sus		6	0	0	0	0	6	0	0	0	6	0	0	0	0	$R^3R^3\times$recessive
3929	3360(2)×2722(2)	res/rty×sus/sus		16	0	16	0	0	16	16	0	0	16	0	0	0	16	$R^3\times$recessive
2632	C.P.C. 2093 Self	res/rty		19	0	9	—	—	—	—	—	—	—	—	—	—	—	R^3
3361	2632(21)×2722(6)	res/rty×sus/sus		12	0	18	0	0	12	18	0	0	12	0	0	0	18	$R^3\times$recessive
2722	C.P.C. 2094 Self	res/rty		12	0	5	0	0	12	5	0	0	12	0	0	0	5	R^3
3348	2722(12)×2024(3)	res/rty×sus/sus		18	0	12	0	0	18	12	0	0	18	0	0	0	12	$R^3\times$recessive
3983	2471(20)×C.P.C. 2092	res/rty×res/rty		—	—	—	0	0	6	0	—	—	—	—	—	—	—	$R^3R^3\times R^3R^3$
4261	3983(1)×2722(9)	res/rty×sus/sus		32	0	0	0	0	32	0	0	0	32	0	0	0	0	$R^3R^3\times$recessive
3988	2471(20)×C.P.C. 2093	res/rty×res/rty		—	—	—	0	0	6	0	—	—	—	—	—	—	—	$R^3R^3\times R^3R^3$
4264	3988(2)×2722(9)	res/rty×sus/sus		—	—	—	0	0	54	0	—	—	—	—	—	—	—	$R^3R^3\times$recessive
3990	2472(26)×2471(16)	res/nec×res/rty		—	—	—	0	6	0	0	—	—	—	—	—	—	—	$R^2R^3\times R^3R^3$
4509	3990(1)×2473(2)	res/nec×sus/sus		32	0	0	0	16	16	0	0	16	16	0	0	0	0	$R^2R^3\times$recessive
4510	3990(2)×2473(2)	res/nec×sus/sus		36	0	0	0	25	11	0	0	25	11	0	0	0	0	$R^2R^3\times$recessive
5623	3990(1)×3993(1)	res/nec×sus/sus		32	0	0	0	17	15	0	0	17	15	0	0	0	0	$R^2R^3\times$recessive
3991	2095(86)×2741(16)	res/res×res/rty		12	0	0	12	0	0	0	12	0	0	0	0	0	0	$R^1\times R^2R^3$
4569	3991(5)×2473(2)	res/res×sus/sus		22	0	10	16	0	6	10	16	0	6	0	0	0	10	$R^1R^3\times$recessive
3356	2652(4)×C.P.C. 2092	res/res×res/rty		6	0	0	—	—	—	—	—	—	—	—	—	—	—	R^1R^1, $R^2R^2\times R^3R^3$
3357	2652(16)×C.P.C. 2092	res/res×res/rty		6	0	0	—	—	—	—	—	—	—	—	—	—	—	R^1R^1, $R^2R^2\times R^3R^3$
3973	3356(1)×2722(6)	res/res×sus/sus		27	0	5	17	0	12	3	17	0	10	0	0	2	3	R^1, R^2, $R^3\times$recessive
3974	3357(6)×2722(6)	res/res×sus/sus		24	0	8	14	0	16	2	14	0	10	0	0	6	2	R^1, R^2, $R^3\times$recessive
4247	3357(6)×2722(9)	res/res×sus/sus		24	0	8	13	0	14	5	13	0	11	0	0	3	5	R^1, R^2, $R^3\times$recessive
3976	2471(16)×C.P.C. 28·4	res/rty×res/res		6	0	0	—	—	—	—	—	—	—	—	—	—	—	$R^2R^3\times R^1R^1$, R^2R^3
4248	3976(1)×2722(9)	res/res×sus/sus		24	0	8	11	0	19	2	11	0	13	0	0	6	2	R^1, R^2, $R^3\times$recessive
4249	3976(2)×2722(9)	res/res×sus/sus		22	0	10	14	0	10	8	14	0	8	0	0	2	8	R^1, R^2, $R^3\times$recessive
2616	C.P.C. 28·4×C.P.C. 2092	res/res×res/rty		6	0	0	—	—	—	—	—	—	—	—	—	—	—	R^1R^1, $R^2R^2\times R^3R^3$
3005	2616(1)×2024(3)	res/res×sus/sus		43	0	17	30	0	22	8	30	0	13	0	0	9	8	R^1, R^2, $R^3\times$recessive
3205	3005(8)×2024(3)	res/res×sus/sus		20	0	9	10	0	14	5	10	0	10	0	0	4	5	R^1, R^2, $R^3\times$recessive
3202	3005(5)×2024(3)	res/rty×sus/sus		14	0	16	0	0	20	10	0	0	14	0	0	6	10	R^2, $R^3\times$recessive
2615	C.P.C. 2093×C.P.C. 28·4	res/rty×res/res		27	0	0	27	0	0	0	27	0	0	0	0	0	0	$R^3\times R^1R^1$, R^2R^2
3012	2024(9)×2615(4)	sus×res/res		23	0	6	12	0	14	3	12	0	11	0	0	3	3	R^1, R^2, $R^3\times$recessive
3000	2615(5)×2024(3)	res/res×sus/sus		49	0	41	49	0	21	20	49	0	0	0	0	21	20	R^1, $R^2\times$recessive
2614	C.P.C. 28·4×C.P.C. 2094	res/res×res/rty		19	0	0	19	0	0	0	19	0	0	0	0	0	0	R^1R^1, $R^2R^2\times R^3$
2993	2614(1)×2024(3)	res/res×sus/sus		39	0	6	25	0	18	2	25	0	14	0	0	4	2	R^1, R^2, $R^3\times$recessive
2995	2614(12)×2024(3)	res/res×sus/sus		35	0	5	14	0	11	—	14	0	11	0	0	—	—	R^1, $R^2\times$recessive
2997	2614(18)×2024(3)	res/res×sus/sus		14	0	16	14	0	7	9	14	0	0	0	0	7	9	R^1, $R^2\times$recessive
2996	2614(17)×2024(3)	res/res×sus/sus		14	9	7	14	0	7	9	14	0	0	3	6	4	2	R^1, R^2, $R^3\times$recessive

parents show, in each case, that the phenotype is inherited as a single unit (table 19). Moreover, it is clear from the intercross data that the genes controlling the reaction in C.P.C. 2092 and C.P.C. 2093 are allelic with the gene present in C.P.C. 12 (table 19, progenies 4261 and 4264).

Progeny 3990 brings together the genes R^2 and R^3, the latter from C.P.C. 12. The three F_2 progenies derived from this combination (4509, 4510 and 5623) each show segregation into approximately equal proportions of the parental phenotypes and none other thus indicating an allelic relationship between the genes concerned, namely R^2 and R^3.

The remaining data of table 19 are derived from various combinations of phenotype 1 with phenotype 3. They form a consistent body indicative of the occurrence of the two genes R^1 and R^3 at separate loci and they again reveal the presence of the independent gene R^6 in material derived from C.P.C. 28·4 and P.I. 160226, the latter *via* seedlings 2652(4) and 2652(16).

(iv) *Seedling 2996(24) and C.P.C. 2712, phenotype 5.* (Table 20)

The sole exception to the orderliness of the data of table 19 was found in progeny 2996 in which there appeared unexpectedly nine plants showing lethal necrosis to virus Y of which three also showed non-lethal rusty necrosis to virus A (phenotype 4) and six were susceptible to virus A (phenotype 5). The significance of these phenotypes was not immediately apparent although it was noted that neither of them had occurred in other segregating progenies derived from C.P.C. 2094 or C.P.C. 28·4. In further examination it became evident that the lethal necrotic reaction to virus Y is under the control of a single gene (progenies 4279, 4277 and 4870 of table 20), and subsequent evidence indicates that this gene is at the same *locus* as that, with identical effect, found in *S. stoloniferum* C.P.C. 2712 (progenies 5154, 5144 of table 20). From the latter (progenies 4239, 4507) the gene is symbolised R^5.

The segregation of progeny 4815 into the parental types only shows that the gene R^5 of C.P.C. 2712 is allelic with the gene R^3 of C.P.C. 2092. Thus, from the relationships established, it is possible to suggest that the gene R^5 of seedling 2996(24) appeared as a mutant of gene R^3 which was demonstrably present in its grandparent C.P.C. 2094 (progenies 2722 and 3348) and parental sibs (progenies 2993 and 2995). There is no substantial further evidence to support this suggestion but it is strengthened by the fact that the phenotype is unmistakable and was never found outwith progeny 2996 until C.P.C. 2712 was acquired at a later date as sample 1452 of the Birmingham University Collection (Cockerham, Davidson and Macarthur, 1963).

Progenies 4511, 4512 and 4741 all indicate in their segregation that R^5 is inherited independently of R^1 and R^6 derived from C.P.C. 28·4 and P.I. 160226. This independence of the two genes is further illustrated in progenies 5167, 5210 and 5222 which provide similar information for combinations of R^5 from 2996 and R^6 from P.I. 160226 *via* seedling 3925(32) and between R^5 from C.P.C. 2712 and R^6 from P.I. 160226 and C.P.C. 28·4 *via* seedlings 3921(1) and 3926(3), respectively (see table 16). These latter progenies also indicate that phenotype 4, lethal necrotic reaction to virus Y combined with non-lethal rusty necrotic reaction to virus A, is not conditioned by a single gene but is the result of the juxtaposition of the two genes R^5 and R^6, the latter alone being present in phenotype 6.

From the material examined there is ample evidence of five genes controlling the reactions of *S. stoloniferum* to viruses Y and A. Three *loci* are

TABLE 20

Progenies related to seedling 2996(24) and C.P. 2172 (Hawkes 1452)

Progeny	Parentage	Reaction of parents Y/A	Reaction of parents Y/A	Segregation on infection with Y only res	Y only nec	Y only sus	A only res	A only nec	A only rty	A only sus	Segregation on infection with Y and A Phenotype with Y/A reaction 1 res/res	2 es/nec	3 res/rty	4 nec/rty	5 nec/sus	6 sus/rty	7 sus/sus	Genotype Dominant gene only
4279	2996(24) Self	nec/sus		0	14	8	—	—	—	—	—	—	—	—	—	—	—	$R^5 \times$ recessive
4277	2996(24) × 2722(4)	nec/sus × sus/sus		0	17	9	—	—	—	—	—	—	—	—	—	—	—	$R^5 \times$ recessive
4870	4279(9) × seedling	nec/sus × sus/sus		0	36	0	—	—	—	—	—	—	—	—	—	—	—	$R^5R^5 \times$ recessive
4239	C.P.C. 2712 × 2722(9)	nec/sus × sus/sus		0	32	0	0	0	0	32	0	0	0	0	32	0	0	$R^5R^6 \times$ recessive
4507	4239(18) × 2473(2)	nec/sus × sus/sus		0	18	18	0	0	0	36	0	0	0	0	18	0	18	$R^5 \times$ recessive
4954	C.P.C. 2712 × 2996(24)	nex/sus × nec/sus		0*	10	0	—	—	—	—	—	—	—	—	—	—	—	$R^5R^5 \times R^5$
5154	4954(1) × 3993(1)	nec/sus × sus/sus		0	54	0	—	—	—	—	—	—	—	—	—	—	—	$R^5R^5 \times$ recessive
5155	4954(2) × 3993(1)	nec/sus × sus/sus		0	160	0	—	—	—	—	—	—	—	—	—	—	—	$R^5R^5 \times$ recessive
4472	C.P.C. 2712 × C.P.C. 2092	nec/sus × res/rty		36	0	0	0	0	36	0	0	0	36	0	0	0	0	$R^5R^5 \times R^6R^6$
4815	4462(19) × seedling	res/rty × sus/sus		34	38	0	0	0	21	15	0	0	21	0	15	0	0	$R^6R^6 \times$ recessive
4237	C.P.C. 2712 × C.P.C. 28-4	nec/sus × res/res		6	0	0	—	—	—	—	—	—	—	—	—	—	—	$R^5R^5 \times R^1R^1$, R^6R^6
4511	4237(1) × 2473(2)	res/res × sus/sus		16	12	8	16	0	11	9	16	0	0	7	5	4	4	R^1, R^5, $R^6 \times$ recessive
4512	4237(2) × 2473(2)	res/res × sus/sus		15	11	6	15	0	5	12	15	0	0	2	9	3	3	R^1, R^5, $R^6 \times$ recessive
4464	C.P.C. 2712 × 2562(4)	nec/sus × res/res		18	0	0	—	—	—	—	—	—	—	—	—	—	—	$R^5R^5 \times R^1R^1$, R^6R^6
4741	4464(19) × seedling	res/res × sus/sus		21	15	17	21	0	17	15	21	0	0	9	6	8	9	R^1, R^5, $R^6 \times$ recessive
3943	3005(69) × 2722(6)	sus/rty × sus/sus		0	0	64	0	0	33	31	0	0	0	0	0	33	31	$R^6 \times$ recessive
4955	3925(32) × 2996(24)	sus/rty × nec/sus		0	13	12	0	0	11	14	0	0	0	5	6		6	$R^6 \times R^5$
5167	4955(4) × 3993(1)	nec/rty × sus/sus		0	20	16	0	0	21	15	0	0	0	14	6	7	9	R^5, $R^6 \times$ recessive
4960	3925(1) × C.P.C. 2712	sus/rty × nec/sus		0	10	0	0	0	6	4	0	0	0	6	4	0	0	$R^6 \times R^5R^5$
4961	3926(3) × C.P.C. 2712	sus/rty × nec/sus		0	10	0	0	0	4	6	0	0	0	4	6	0	0	$R^6 \times R^5R^5$
5210	4960(8) × 3993(1)	nec/rty × sus/sus		0	16	20	0	0	19	17	0	0	0	9	7	10	10	R^5, $R^6 \times$ recessive
5222	4961(10) × 3993(1)	nec/rty × sus/sus		0	14	22	0	0	20	16	0	0	0	8	6	12	10	R^5, $R^6 \times$ recessive

concerned. At the first *locus* is the gene R^1 which controls extreme resistance to both viruses and conditions phenotype 1. At the second *locus* there are three genes R^2, R^3 and R^5 which condition phenotypes 2, 3 and 5, respectively, and at the third *locus* there is the gene R^6 which modifies reaction to virus A in plants which are basically susceptible to both viruses and gives rise to phenotype 6 alone and to phenotype 4 in association with R^5. The order of epistasis and dominance is $R^1 > R^2 > R^3 > R^5 > r$ in relation to virus Y and $R^1 > R^2 > R^3 > R^6 > r$ in relation to virus A.

(v) *Relationships between the genes* Ry, Ryn, Rym (*Ross, 1960*) *and* R^1, R^2, R^3 R^5 *and* R^6. (Table 21)

Ross (1960) postulated three genes, *Ry*, *Ryn*, *Rym*, to explain the inheritance of reactions to viruses Y and A in *S. stoloniferum*. From his description of the joint reactions to the two viruses, gene *Ry* is similar in effect to the R^1 of the present report and controls reactions of phenotype 1. *Ryn*, on the other hand, is reported to control lethal necrotic (hypersensitive) responses to virus Y and local lesion (hypersensitive) responses to virus A followed, in the latter case only, by systemic invasion and mosaic symptoms. In our material no phenotype of this kind has been observed. *Rym* is in control of a similar reaction to that recorded herein as due to the gene R^6, *i.e.* phenotype 6. Direct comparisons were made through selected inter-crosses between our material and material supplied by Dr Ross. The segregation details are given in table 21.

Seed sample R58/141 produced 29 plants of which 21 were phenotype 1 and eight were phenotype 5 when tested. There were no plants susceptible to both virus Y and virus A. Twenty of the phenotype 1 plants were mated with recessive seedlings and pilot tests with virus Y were made on samples of the progenies. Ten progenies showed no segregation and ten progenies segregated into resistant and lethal necrotic types. A large progeny of the non-segregating and two large progenies of the segregating types were then tested with virus Y. The first, progeny 4578, contained 131 resistant : 1 susceptible whereas progenies 4583 and 4590 contained approximately equal numbers of resistant and necrotic types with a single susceptible plant in each.

The main indication from the above data is that the original seed was obtained from a self-fertilised plant possessing the genes *Ry* and *Ryn* and from the further details, ignoring the anomalous susceptible plants, it is clear that *Ry* and *Ryn* are allelic genes. The exceptional appearance of susceptible plants, repeated again in progenies 4738 and 4819, was unexpected in an acknowledged allotetraploid species but the similarity of their occurrence to those found in admittedly autotetraploid material (section (b)) may denote that the genes *Ry* and *Ryn* are situated on chromosomes which occasionally pair with related chromosomes of the alternative genome. Observations on quadrivalent formation in some forms of *S. stoloniferum* may be interpreted as evidence of the homologies necessary for such pairing (Gilles, 1955).

The genes *Ry* and R^1 were brought together in the cross 4448 from which two F_2 progenies (4737 and 4738) were raised and examined. In the first there was no segregation and in the second 35 plants were of phenotype 1 while one plant was susceptible, a result which points to the allelic relationship of the two genes and which was confirmed by a similar association between *Ryn* and R^1 in progenies 4818 and 4819.

Progenies 4827, 4820 and 4822 show that R^2 was inherited independently

TABLE 21

Progenies showing relationship with R^1, R^3, R^5, R^6 and R^8 with Ry, Ryn, Rym (*Ross*, 1960)

Progeny	Parentage	Parental genotypes Dominant genes only	Reactions of parents Y/A	Y/A	Segregation on infection with Y only res	nec	sus	A only res	nec	rty	sus	Segregation on infection with Y and A Phenotypes with Y/A reaction 1 res/res	2 res/nec	3 res/rty	4 nec/rty	5 nec/sus	6 sus/rty	7 sus/sus	Indication
58/141	—	*RyRyn*	—		21	8	0	21	0	0	8	21	0	0	0	8	0	0	*RyRyn* self
4578	58/141/5 × 2743(2)	*RyRy* × recessive	res/res × sus/sus		131	0	1	—	—	—	—	—	—	—	—	—	—	—	*Ryn* allelic with *Ry*
4583	58/141/21 × 2473(2)	*RyRyn* × recessive	res/res × sus/sus		50	52	1	—	—	—	—	—	—	—	—	—	—	—	*Ryn* allelic with *Ry*
4590	58/141/36 × 2473(2)	*RyRyn* × recessive	res/res × sus/sus		83	102	1	—	—	—	—	—	—	—	—	—	—	—	*Ryn* allelic with *Ry*
58/139	—	*RynRyn*	nec/sus		0	32	0	0	0	0	32	0	0	0	0	32	0	0	*RynRyn* self
4448	58/141/5 × C.P.C. 28·4	$RyRy \times R^1R^1$, R^6R^6	res/res × res/res		2	0	0	—	—	—	—	—	—	—	—	—	—	—	
4737	4448(1) × seedling	RyR^1, R^6 × recessive	res/res × sus/sus		—	—	—	154	0	0	0	—	—	—	—	—	—	—	R^1 allelic with *Ry*
4738	4448(2) × seedling	RyR^1, R^6 × recessive	res/res × sus/sus		35	0	1	135	0	0	2	135	0	0	0	0	0	2	R^1 allelic with *Ry*
4447	58/139/9 × C.P.C. 28·4	$RynRyn \times R^1R^1$, R^6R^6	nec/sus × res/res		2	0	0	—	—	—	—	—	—	—	—	—	—	—	
4818	4447(1) × seedling	R^1Ryn, R^6 × recessive	res/res × sus/sus		22	26	2	22	0	10	8	22	0	0	10	6	0	2	R^1 allelic with *Ryn*
4819	4447(2) × seedling	R^1Ryn, R^6 × recessive	res/res × sus/sus		32	31	1	32	0	12	18	32	0	0	12	17	0	1	R^6 independent
4460	58/141/38 × 2472(4)	$RyRy \times R^3R^3$, R^6R^6	res/res × res/nec		2	0	0	—	—	—	—	—	—	—	—	—	—	—	
4287	4460(1) × 4276(2)	*Ry*, R^3, R^6 × recessive	res/res × sus/sus		64	0	22	44	20	13	9	44	20	0	0	0	13	9	R^3 independent of *Ry* and R^6
4457	58/139/12 × 2472(4)	$RynRyn \times R^3R^3$, R^6R^6	nec/sus × res/nec		2	0	0	—	—	—	—	—	—	—	—	—	—	—	
4820	4457(1) × 4276(2)	R^3, *Ryn*, R^6 × recessive	res/nec × sus/sus		25	12	12	0	20	13	7	0	20	0	5	5	8	2	R^3 independent of
4822	4457(2) × seedling	R^3, *Ryn*, R^6 × recessive	res/nec × sus/sus		48	19	23	0	30	15	9	0	30	0	10	4	5	5	*Ryn* and R^6
4467	58/141/25 × 2471(16)	$RyRy \times R^5R^5$	res/res × res/rty		—	—	—	2	0	0	0	—	—	—	—	—	—	—	
4745	4467(1) × seedling	*Ry*, R^5 × recessive	res/res × sus/sus		—	—	—	19	0	8	9	—	—	—	—	—	—	—	R^5 independent of
4465	58/139/11 × 2471(16)	$RynRyn \times R^5R^5$	nec/sus × res/rty		2	0	0	—	—	—	—	—	—	—	—	—	—	—	*Ry* and *Ryn*
4825	4465(1) × seedling	*Ryn*, R^5 × recessive	res/rty × sus/sus		19	5	12	—	—	—	—	—	—	—	—	—	—	—	
4475	58/141/9 × C.P.C. 2092	$RyRy \times R^5R^5$	res/res × res/rty		—	—	—	2	0	0	0	—	—	—	—	—	—	—	
4752	4475(1) × seedling	*Ry*, R^5 × recessive	res/res × sus/sus		—	—	—	26	0	6	4	—	—	—	—	—	—	—	
5631	4475(2) × 3993(1)	*Ry*, R^5 × recessive	res/res × sus/sus		37	0	12	22	0	15	12	22	0	15	0	0	0	12	R^5 independent of
4473	58/139/14 × C.P.C. 2092	$RynRyn \times R^5R^5$	nec/sus × res/rty		2	0	0	—	—	—	—	—	—	—	—	—	—	—	*Ry* and *Ryn*
4749	4473(2) × seedling	R^5, *Ryn* × recessive	res/rty × sus/sus		19	7	10	—	—	—	—	—	—	—	—	—	—	—	
5630	4473(1) × 3993(1)	R^5, *Ryn* × recessive	res/rty × sus/sus		28	12	10	0	0	27	15	0	0	27	0	8	0	7	
4952	58/141/21 × 2996(24)	*RyRyn* × R^5	res/res × nec/sus		5	5	0	—	—	—	—	—	—	—	—	—	—	—	
5142	4952(9) × 3993(1)	*Ry*, R^5 × recessive	res/res × sus/sus		0	153	55	—	—	—	—	—	—	—	—	—	—	—	
5138	4952(5) × 3993(1)	*Ryn*, R^5 × recessive	res/res × sus/sus		26	16	16	—	—	—	—	—	—	—	—	—	—	—	R^5 independent of
4953	58/139/33 × 2996(24)	*RynRyn* × R^5	nec/res × nec/sus		0	10	0	—	—	—	—	—	—	—	—	—	—	—	*Ry* and *Ryn*
5146	4953(3) × 3993(1)	*Ryn*, R^5 × recessive	nec/sus × sus/sus		0	156	50	—	—	—	—	—	—	—	—	—	—	—	
5147	4953(4) × 3993(1)	*Ryn*, R^5 × recessive	nec/sus × sus/sus		0	39	15	—	—	—	—	—	—	—	—	—	—	—	
4497	58/141/44 × 3926(3)	*RyRy* × R^6	res/res × sus/rty		—	—	—	10	0	0	0	—	—	—	—	—	—	—	
4805	4497(2) × seedling	*Ry*, R^6 × recessive	res/res × sus/sus		—	—	—	18	0	9	9	—	—	—	—	—	—	—	R^6 independent of
4493	58/139/34 × 3925(27)	*RynRyn* × R^6	nec/sus × sus/rty		0	10	0	0	0	3	2	0	0	0	3	2	0	0	*Ry* and *Ryn*
4933	4493(5) × seedling	*Ryn*, R^6 × recessive	nec/rty × sus/sus		0	16	16	0	0	14	17	0	0	0	3	13	11	4	
5635	59/189/24 × 3993(1)	*Ry*, *Rym* × recessive	res/res × sus/sus		—	—	—	42	0	22	20	—	—	—	—	—	—	—	*Rym* independent of *Ry*
4495	59/192/2 × 3925(27)	*Rym* × R^6	sus/rty × sus/rty		—	—	—	0	0	18	7	—	—	—	—	—	—	—	
4900	4495(3) × seedling	$RymR^6$ × recessive	sus/rty × sus/sus		—	—	—	0	0	46	0	—	—	—	—	—	—	—	R^6 allelic with *Rym*
4905	4495(2) × seedling	$RymR^6$ × recessive	sus/rty × sus/sus		—	—	—	0	0	49	0	—	—	—	—	—	—	—	
4959	51/191/2 × C P.C. 2712	*Rym* × R^5R^5	sus/rty × nec/sus		0	10	0	0	0	6	4	0	0	0	6	4	0	0	R^5 independent of *Rym*
5198	4959(6) × 3993(1)	R^5, *Rym* × recessive	nec/rty × sus/sus		0	22	28		0	22	14	0	0	0	8	7	14	7	

of *Ry* and *Ryn* as were its alleles R^3 and R^5 (progenies 4745, 4825, 4752, 5631, 4749, 5630 and 5138, 5142, 5146, 5147, respectively). Progenies 4805, 4933 and 5635 indicate that *Ry* and *Ryn* are inherited independently of R^6 and *Rym* whereas progenies 4900 and 4905 point to these latter genes being alleles.

The main features of interest in these data are found associated with the gene *Ryn*. According to Ross (1960) this gene controls a total necrotic response to virus Y and a local necrotic response to virus A which is followed by systemic invasion and mosaic symptoms. Confirmation was obtained of the lethal reaction to virus Y but towards virus A we observed no necrosis and material containing the gene *Ryn* only behaved as susceptible to the virus thus giving rise to phenotype 5 in the same fashion as the gene R^5. The most probable explanation of this difference in recorded reaction is that Ross's source of virus A was contaminated with virus Y. Such a mixture would give rise to the phenotype which he ascribes to the *Ryn* gene. Mixed infections of virus A and virus Y are not uncommon and to safeguard against them it is our practice to culture virus A in *S. stoloniferum* seedlings of phenotype 5 homozygous for the gene R^5 which are protected by hypersensitivity from the Y virus. Similarly, we culture virus Y in seedlings of phenotype 6 homozygous for the gene R^6 which indicate the presence of virus A with a non-lethal but highly necrotic reaction.

From our observations, material with the gene *Ryn* is indistinguishable from that with the gene R^5, yet from the results of genetic comparison *Ryn* is situated at the *locus* carrying the gene *Ry* which gives rise to phenotype 1 indistinguishable from that controlled by R^1 at the same *locus*. R^5, on the other hand, is at a second *locus* already shown to be the site of R^2 and R^3. The question arises, therefore, as to whether we are dealing with two similar genes at different *loci* or whether the genes are identical but are situated at corresponding *loci* in the two genomes of an allotetraploid. The indirect evidence of quadrivalent formation in *S. stoloniferum* (Gilles, 1955) and the segregation data of Ross (1958) are suggestive of the latter alternative and further support was obtained in progeny 4251, an F_2 family from 2472 (26), $R^2R^2 : R^6(R^6) \times$ C.P.C. 28·4, $R^1R^1 : R^6R^6$ which segregated entirely into the parental types with 33 seedlings of phenotype 1 (R^1) and 35 seedlings of phenotype 2 (R^2) thus indicating that R^1 and R^2 are alleles. Progenies derived from a sister F_1 plant (progenies 4250, 5604, table 18), on the other hand, showed full segregation into phenotypes 1, 2 and 6 indicative of independence of R^1 and R^2. It seems not improbable that chance pairing of corresponding chromosomes from the two genomes gave rise to the F_1 parent of progeny 4251 and that the more usual pairing within the genome provided the parent of progenies 4250 and 5604. Pairing within the genome is considered to be the more usual because of the evidence of allelism between R^2 and R^3 shown in table 18 (progenies 4509, 4510, and 5623).

If the two *loci* of R^1, *Ry* (R^2), *Ryn* and R^2, R^3, R^5 are indeed parentally identical then the genes controlling reaction to viruses Y and A within *S. stoloniferum* are $R^1(Ry) > R^2 > R^3 > R^5(Ryn > r$ in descending order of dominance with the gene $R^6(Rym)$ at an independent *locus* and controlling a minor reaction to virus A only.

5. Discussion

Throughout this paper, and for reasons which are apparent, gene symbols from earlier investigations have been used wherever possible. These symbols

are neither uniform nor sufficiently discriminating in distinguishing genes with similar effect but located in different species or at different *loci*. It is suggested, therefore, that they be re-symbolised in conventional manner to indicate their basic type, origin and effect. Thus the genes which control reaction to virus X may be symbolised as follows:

Nx$_{tbr}$, in cultivars of *S. tuberosum* controls a lethally necrotic (hypersensitive) reaction to strains of virus X within Groups 1 and 3 (Cockerham, 1954). It is identical with Nx (Cadman, 1942) and is linked to Na$_{tbr}$ (Na, Cadman, 1942) and possibly to Nc$_{tbr}$ (Nc, Cadman, 1942) but is independent of Nb$_{tbr}$ (Nb, Cadman, 1942), Rx$_{adg}$ and Rx$_{acl}$ (X^1, Cockerham, 1958). The gene probably occurs also in *S. andigena*, C.P.C. 65, 66, 91A, 102, 106, 146, 203, 204 and 236.

Nx$_{tbr}$spl, in *S. sparsipilum*, controls lethal necrosis to virus X, Groups 1 and 3. It is allelic and probably identical with Nx$_{tbr}$ and is linked to Nx$_{chc}$ and Ny$_{chc}$.

Nb$_{tbr}$, in *S. tuberosum* (cultivars), controls lethal necrosis to virus X, Groups 1 and 2. It is identical with Nb (Cadman, 1942) but is independent of Nx$_{tbr}$, Na$_{tbr}$, Nc$_{tbr}$ and Rx$_{acl}^n$ (X^n, Cockerham, 1958). Probably it occurs also in *S. andigena*, C.P.C. 91B, 130, 188, 218, and 264.

Nx$_{chc}$, in *S. chacoense*, controls lethal necrosis to all strains of virus X. It is linked to Nx$_{tbr}^{spl}$ and Ny$_{chc}$. The same gene or an allele occurs also in *S. microdontum*.

Rx$_{adg}$, in *S. andigena* and U.S. seedling 41956, controls extreme resistance to all strains of virus X. It is independent of Nx$_{tbr}$ and Rx$_{acl}$.

Rx$_{acl}$, in *S. acaule*, controls extreme resistance to all strains of virus X. It is probably identical with X^1 (Cockerham, 1958) but is independent of Nx$_{tbr}$ and Rx$_{adg}$.

Rx$_{acl}^n$, an allele of Rx$_{acl}$, is found in *S. andigena* and controls lethal necrosis to all strains of virus X. It is probably identical with X^n (Cockerham, 1958) but is independent of Nb$_{tbr}$.

The genes relating to the Y group of viruses may be symbolised similarly as follows:

Na$_{tbr}$, in *S. tuberosum*, controls lethal necrosis to virus A. It is identical with Na (Cadman, 1942) and is linked to Nx$_{tbr}$ and possibly to Nc$_{tbr}$. It is independent of Nb$_{tbr}$. It may occur also in *S. andigena* C.P.C. 56, 58, 61, 69, 87, 106, 110, 130, 132, 138, 141, 142, 146, 147, 150, 155, 182, 188, 191, 204, 210, 225, 236, 239, 244, 250, 274, 278.

Nc$_{tbr}$, in *S. tuberosum*, controls lethal necrosis to virus strain Y^c. It is identical with Nc (Cadman, 1942) and possibly linked to Nx$_{tbr}$ and Na$_{tbr}$. It may occur also in *S. andigena* C.P.C. 67, 102, 136, 141, 183, 203, 207, 228, 278.

Ny$_{chc}$, in *S. chacoense* and *S. microdontum*, controls lethal necrosis to all strains of virus Y and virus A. It is linked to Nx$_{chc}$ and Nx$_{tbr}^{spl}$.

Ny$_{dms}$, in *S. demissum*, controls lethal necrosis to all strains of virus Y and virus A. It is identical with Ny (Cockerham, 1958) but is independent of Ry$_{hou}$.

Ry$_{dms}^a$, an allele of Ny$_{dms}$, controls lethal necrosis to virus A. It is identical with Na (Cockerham, 1958).

Ry$_{hou}$, in *S. hougasii*, controls extreme resistance to all strains of virus Y and virus A. It is independent of Ny$_{dms}$.

Ry_{sto}, in *S. stoloniferum*, controls extreme resistance to all strains of virus Y and virus A. It is identical with *Ry* (Ross, 1960) and R^1 of section (e) above.

Ry_{sto}^{na}, in *S. stoloniferum*, controls extreme resistance to virus Y and lethal necrosis to virus A. It is a probable allele of Ry_{sto} and a confirmed allele of Ry_{sto}^{rna}. It is designated R^2 in section (e) above.

Ry_{sto}^{n1}, in *S. stoloniferum*, controls lethal necrosis to virus Y coupled with susceptibility to virus A. It is identical with *Ryn* (Ross, 1960) and is an allele of Ry_{sto}. Only independent relationships have been found with Ry_{sto}^{rna}, Ry_{sto}^{n2} and Na_{sto}.

Ry_{sto}^{rna}, in *S. stoloniferum*, controls extreme resistance to virus Y and non-lethal rusty necrosis to virus A. It is an allele of Ry_{sto}^{na} and Ry_{sto}^{n2} but has been found to be independent of Ry_{sto}, Ry_{sto}^{n1} and Na_{sto}. It is designated R^3 in section (e) above.

Ry_{sto}^{n2}, in *S. stoloniferum*, is identical in effect with Ry_{sto}^{n1} but is independent of the latter and its allele Ry_{sto}. It is also independent of Na_{sto}. It is an allele of Ry_{sto}^{rna} and is R^5 of section (e) above.

Na_{sto}, in *S. stoloniferum*, controls a non-lethal rusty necrosis to virus A. It is designated R^6 in section (e) above and is identical with *Rym* (Ross, 1960) but is independent of the Ry_{sto} and Ry_{sto}^{rna} *loci*.

Our understanding of the pattern of gene relationships, even within the few species examined, is still incomplete and there are other species yet to investigate. The evidence available, however, indicates that reactions to virus X are conditioned by six genes situated at four, or possibly five, different *loci*. (There are no data on the relationship of Rx_{and} with Nb_{tbr}). Two of these *loci*, Nx_{tbr} and Nx_{chc}, appear to be associated in a linkage group which also contains the *loci* of genes controlling reactions to Y viruses, namely, Na_{tbr}, Ny_{chc} and, possibly, Nc_{tbr}. A significant feature of this association is that each of the genes concerned controls a lethal necrotic reaction and it may well be that the whole group of genes is situated within a section of chromosome which is in overall control of this response to infection irrespective of the virus evoking the response. It is rather surprising, therefore, to find that the gene Nb_{tbr}, which conditions a similar reaction, is completely independent. The allelic genes of *S. demissum* also control lethal necrotic reactions but there are no data upon which to examine their relationships with the other genes of similar action either to virus Y, which actuates them, or to virus X. It is established, however, that they are unrelated to the gene Ry_{hou} of *S. hougasii*.

The genes controlling the various reactions of *S. stoloniferum* to virus Y and the related virus A show anomalies of relationship. Three *loci* are concerned of which one is completely independent and is the site of the relatively unimportant gene Na_{sto}. In most of the comparisons made, the other two *loci* appear to be independent of each other but the occurrence of distinctive similar genes, Ry_{sto}^{n1}, and Ry_{sto}^{n2} at each *locus*, coupled with the evidence of allelic relationships between Ry_{sto}^{na} and genes at both *loci*, has indicated a relationship between the two *loci*. The interpretation offered, for which there is supporting evidence, is that the two *loci* are in corresponding genomes of an allotetraploid which occasionally functions, partially at least, as an autotetraploid. This interpretation may have significance in evolutionary studies as also may the evidence of introgression

between species provided by the identity of the genes controlling reaction to virus Y in *S. chacoense* and *S. microdontum* (cf. Hawkes and Hjerting, 1969) and the identification of the gene controlling necrotic response to all strains of virus X in *S. andigena* as an allele of the gene controlling extreme resistance to the same virus in *S. acaule*. Evolutionary significance may also be attached to the allelic relationship of the genes controlling lethal necrotic response to virus X, groups 1 and 3, in *S. tuberosum* and *S. sparsipilum* since it offers a direct connection between the wild, diploid species and the cultivated tetraploid species of the series Tuberosa.

6. Summary

1. The genetic control of extreme resistance and necrotic (hypersensitive) reaction to potato virus X and potato virus Y in several species of tuber-bearing Solanaceae is demonstrated with emphasis on relationships between some of the genes concerned.

2. Seven genes controlling reaction to virus X are recorded. Using a suggested uniform symbolism these are Nx_{tbr} of *S. tuberosum* and its allele $Nx_{tbr}{}^{spl}$ of *S. sparsipilum* which control necrotic reactions to X—viruses of Groups 1 and 3 and Nb_{tbr}, an independent gene of *S. tuberosum*, which controls necrotic reaction to the X—viruses of Groups 1 and 2; Nx_{chc} which, in *S. chacoense* and *S. microdontum*, controls necrotic reaction to all strains of virus X and which, in hybrids with *S. sparsipilum*, is linked to $Nx_{tbr}{}^{spl}$; Rx_{adg} of *S. andigena* and Rx_{acl} of *S. acaule* which are independent genes controlling extreme resistance to virus X and $Rx_{acl}{}^{n}$ which is an allele of the latter found in both *S. andigena* and *S. acaule* in which it controls necrotic reaction to the whole virus.

3. With regard to virus Y twelve genes are recorded. Two of the occur in *S. tuberosum* where Na_{tbr} and Nc_{tbr} control necrotic reactions to viruses A and C respectively, both viruses being part of the virus Y complex. Na_{tbr} is closely linked to Nx_{tbr} and there is evidence, though anomalies occur, that Nc_{tbr} is in the same linkage group.

4. Ny_{chc} occurs in *S. chacoense* and *S. microdontum* and is activated by all strains of virus Y. It is linked to Nx_{chc}.

5. Ry_{hou} in *S. hougasii* controls extreme resistance to the whole of virus Y. It is independent of Ny_{dms} which, in the related species *S. demissum*, controls necrotic reaction to virus Y. Na_{dms} is an allele of the latter controlling necrotic reaction to virus A only.

6. The bulk of evidence from *S. stoloniferum* indicates that various phenotypes observed in reaction to viruses Y and A are determined by six genes of which Ry_{sto} and $Ry_{sto}{}^{n1}$ are at one *locus*, $Ry_{sto}{}^{na}$, $Ry_{sto}{}^{rna}$ and $Ry_{sto}{}^{n2}$ are at a second *locus* and Na_{sto} is at a third *locus*. Supplementary evidence, however, is sufficiently strong to suggest that the first two *loci* are replicates within the two genomes of an allotetraploid which shows occasional homoeologous recombination. In this case the genes $Ry_{sto}{}^{n1}$ and $Ry_{sto}{}^{n2}$ may be identical.

7. The significance of the gene relationships to evolutionary studies is discussed briefly.

7. References

BENSON, A. P., AND HOOKER, W. J. 1960*a*. Isolation of virus X from " immune " varieties of potato, *Solanum tuberosum*. *Phytopath.*, *50*, 629.

BENSON, A. P., AND HOOKER, W. J. 1960*b*. Carbohydrate reserves in grafted plants of potato varieties resistant to virus X. *Phytopath.*, *50*, 629.

BLACK, W. 1956. Breeding potatoes for resistance to virus and other agents of disease. *Genetica Agraria.*, *7*, 1-2.

CADMAN, C. H. 1942. Autotetraploid inheritance in the potato: some new evidence. *J. Genet.*, *44*, 33-52.

COCKERHAM, G. 1939. The distribution and significance of certain potato viruses in Scotland. *Scot. J. Agric.*, *22*, 1-11.

COCKERHAM, G. 1943*a*. Potato breeding for virus resistance. *Ann. appl. Biol.*, *30*, 105-108.

COCKERHAM, G. 1943*b*. The reactions of potato varieties to viruses X, A, B and C. *Ann. appl. Biol.*, *30*, 338-344.

COCKERHAM, G. 1945. Some genetical aspects of resistance to potato viruses. *Ann. appl. Biol.*, *32*, 280-281.

COCKERHAM, G. 1952. Breeding for resistance to potato viruses. *Proc. Conf. Pot. Virus Dis., Wageningen*, 37-39.

COCKERHAM, G. 1954. Strains of potato virus X. *Proc. 2nd Conf. Virus Dis., Lisse-Wageningen*, 25-29.

COCKERHAM, G. 1958. Experimental breeding in relation to virus resistance. *Proc. 3rd Conf. Pot. Virus Dis., Lisse-Wageningen*, 199-203.

COCKERHAM, G. 1962. The status of the X^d strain of potato virus X. *Scott. Plant Breeding Statn. Record*, 39-48.

COCKERHAM, G., AND DAVIDSON, T. M. W. 1963. Note on an unusual strain of potato virus X. *Scott. Plant Breeding Statn. Record*, 26-29.

COCKERHAM, G., DAVIDSON, T. M. W., and MACARTHUR, A. W. 1963. Report on the virus content and the reactions to viruses X, S and Y of tuber-bearing solanums collecetd by the Birmingham University Expeditions to Mexico and Central America, 1958. *Scott. Plant Breeding Statn. Record*, 30-34.

DUNNETT, J. M. 1957. Variation in pathogenicity of the potato-root eelworm (*Heterodcra rostochiensis* Wall.) and its significance in potato breeding. *Euphytica*, *6*, 77-89.

EASTON, G. D., LARSON, R. H., AND HOUGAS, R. W. 1958. Immunity to virus Y in the genus *Solanum*. *Univ. Wisc. Res. Bull.*, 205.

GANGULY, A. 1949. Inheritance studies on 24 chromosome tuber-bearing *Solanum* species. *Thesis. Univ. Edinburgh.*

GILLES, A. 1955. Recherches cytogénétiques sur les *Solanum* (Section Tuberarium). I. Nombres chromosomiques et associations méitoiques. *Cellule, Louvain*, *57*, 7-31.

HAWKES, J. G. 1945. The indigenous American potatoes and their value in plant breeding. *Empire J. Agric.*, *13*, 11-40.

HAWKES, J. G. 1963. A revision of the tuber-bearing solanums. *Scott. Plant Breeding Statn. Record*, 76-181.

HAWKES, J. G., AND HJERTING, J. P. 1969. The potatoes of Argentina, Brazil, Paraguay and Uruguay. *Anns. of Bot. Memoirs No. 3, London.*

HOUGAS, R. W., AND PELOQUIN, S. J. 1957. A haploid plant of the potato variety Katahdin. *Nature*, *180*, 1209-1210.

HOUGAS, R. W., AND PELOQUIN, S. J. 1958*a*. The potential of potato haploids in breeding and genetic research. *Amer. Pot. J.*, *35*, 701-707.

HOUGAS, R.W., PELOQUIN, S. J., AND ROSS, R. W. 1958*b*. Haploids of the common potato. *J. Hered.*, *49*, 103-106.

HOUGAS, R. W., AND PELOQUIN, S. J. 1960. Crossability of *Solanum tuberosum* haploids with diploid *Solanum* species. *Eur. Potato J.*, *3*, 325-330.

HOWARD, H. W., AND FULLER, J. M. 1965. The inheritance of top-necrosis to viruses X, A, B and C in *Solanum tuberosum*. *Euphytica*, *14*, 189-195.

HUTTON, E. M., AND WARK, D. C. 1952. A relationship between immunity and localized reaction to virus X in the potato (*Solanum tuberosum*). *Austr. J. Sci. Res.*, *5*, 237-243.

MILLS, W. R. 1965. Inheritance of immunity to potato virus X. *Amer. Pot. J.*, *42*, 294-295.

ROSS, H. 1952. Studies on mosaic resistance in the potato. *Proc. Conf. Pot. Virus, Dis., Wageningen*, 40-47.

ROSS, H. 1954*a*. Über die extreme resistenz von *Solanum acuale* gegen das X-virus. *Mitt. Biol. Bund. Berlin-Dahlem*, 80.

ROSS, H. 1954*b*. Die Vererbung der "Immunitat" gegen das X-virus in tetraploidem *Solanum acaule*. *Carylogia* 6 (suppl.), 1128-1132.

ROSS, H. 1958. Inheritance of extreme resistance to virus Y in *Solanum stoloniferum* and its hybrids with *Solanum tuberosum*. *Proc. 3rd Conf. Pot. Virus Dis., Lisse-Wageningen*, 204-211.

ROSS, H. 1960. Über die vererbung von eigenschaften fur resistenz gegen das Y- und A-virus in *Solanum stoloniferum* und die mogliche bedeutung fur eine allgemeine genetik der virus resistenz in *Solanum* sect. *Tuberarium*. *Proc. 4th Conf. Pot. Virus Dis., Braunschweig*, 40-49.

ROSS, H., AND BAERECKE, M. L. 1950. III. Selection for resistance to mosaic virus (diseases) in wild species and in hybrids of wild species of potatoes. *Amer. Pot. J., 27*, No. 8, 275-284.

SALAMAN, R. N. 1938. The potato virus X: its strains and reactions. *Phil. Trans. Roy. Soc. London*, B.299, 137-217.

SCHULTZ, E. S., AND RALEIGH, W. P. 1933. Resistance of potato to latent mosaic. (Abstract) *Phytopath, 23*, 32.

STEVENSON, F. J., SCHULTZ, E. S., AND CLARK, C. F. 1939. Inheritance of immunity from virus X (latent mosaic) in the potato. *Phytopath, 29*, 362-365.

WIERSEMA, H. T. 1961. Methods and means used in breeding potatoes with extreme resistance to viruses X and Y. *Proc. 4th Conf. Pot. Virus Dis., Braunschweig*, 30-36.