

INHERITANCE OF SPOTTED WILT RESISTANCE IN THE TOMATO

II. FIVE GENES CONTROLLING SPOTTED WILT RESISTANCE IN FOUR TOMATO TYPES

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Summary

The four spotted-wilt-resistant tomato types, *Lycopersicon pimpinellifolium* and *L. esculentum* varieties Rey de los Tempranos, Pearl Harbour, and Manzana, were each crossed with the susceptible variety Potentate, and also crossed with each other in all possible combinations. The F₁ and F₂ plants, when inoculated with 10 strains of tomato spotted wilt virus, were resistant to five groups of these strains. Three independently inherited recessive genes and two dominant alleles were shown to control resistance to the five groups of spotted wilt strains in the resistant tomato types. Previous evidence of synergism of spotted wilt strains was substantiated by experimental results.

The relative value of each of the four resistant tomato types is discussed together with the best method of utilizing their resistance in practical breeding programmes. A method for selecting resistant phenotypes in hybrid progenies is suggested.

I. INTRODUCTION

Many attempts have been made to breed commercial tomato varieties resistant to tomato spotted wilt (T.S.W.) virus. Porter's strain of *Lycopersicon pimpinellifolium* was used by Kikuta, Hendrix, and Frazier (1945) to breed the variety Pearl Harbour, which was resistant to T.S.W. in Hawaii and appeared to possess a single dominant gene controlling resistance.

Hutton and Peak (1949) used Porter's strain of *L. pimpinellifolium* to breed T.S.W.-resistant hybrids with medium-sized fruit. They reported the inheritance of resistance as being obscure but likely to be controlled by a polygenic system.

Holmes (1948) found that two Argentinian varieties of *L. esculentum*, Rey de los Tempranos and Manzana, were resistant to T.S.W. in New Jersey. Resistance in Rey de los Tempranos appeared to be controlled by a single recessive gene. Holmes concluded that this gene for resistance may be treated as though it were fully recessive in breeding experiments, although F₁ heterozygotes were shown to be more difficult to infect systemically than were the susceptible parent plants. Both Rey de los Tempranos and Manzana were found to be susceptible to T.S.W. in most other areas of the world.

Finlay (1951) reported that the F₁ hybrid of a cross between Pearl Harbour and Rey de los Tempranos had very high field resistance to the disease. The partial resistances of both parents were apparently additive in the F₁ progeny.

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Reporting the results of 12 years of testing resistance of tomato hybrids to natural infection of the T.S.W. virus in field plots, Smith and Gardner (1951) noted that the level of resistance found in Porter's strain of *L. pimpinellifolium* had not been recovered in any of its progeny following a cross with a susceptible variety. No evidence of simple Mendelian inheritance of resistance was obtained.

In all these breeding programmes the resistance or susceptibility of segregating phenotypes was assessed by their reaction to either infection with the disease in the field, or to a natural complex of the virus strains mechanically inoculated. This approach has led to a meagre and confused understanding of the inheritance mechanism of T.S.W. resistance in tomatoes.

Hutton and Peak (1952) have shown that the efficiency of the T.S.W. virus-inactivating system in some resistant tomato species varies considerably with changes in temperature. They suggest that inoculation of hybrid progenies with the ringspot strain of T.S.W. at a constant temperature of 90°F. may facilitate the selection of resistant and susceptible phenotypes.

Finlay (1952) recorded the resistance or susceptibility of some tomato species to 10 strains of the T.S.W. virus under a fixed set of environmental conditions. The present paper records the results of experiments in which the resistance or susceptibility of tomato species and varieties to 10 strains of T.S.W. was used to elucidate the number and mode of inheritance of genes controlling T.S.W. resistance.

II. MATERIALS AND METHODS

(a) Preparation of Genetic Material

(i) *Parent Varieties and Species*.—The tomato varieties and species selected for the study of the genetics of T.S.W. resistance were Porter's strain of *L. pimpinellifolium* and *L. esculentum* varieties Rey de los Tempranos, Pearl Harbour, and Manzana. All these types have some form of resistance to T.S.W. The variety Potentate was included as the susceptible control. *L. peruvianum* was excluded from this study because of its cross-sterility with the above-mentioned tomato types.

(ii) *F₁ and F₂ Hybrid Populations*.—The F₁ and F₂ plants required for the first experiments to identify genes for resistance to T.S.W. were obtained by crossing each of the four resistant tomato types with the susceptible variety Potentate. Selfing of some of the F₁ plants produced seed for the F₂ populations.

In the second experiment designed to test allelism, the four resistant types were crossed with each other in all possible combinations. Seed to produce F₁ and F₂ populations was maintained until required.

The parent plants used for these crosses were grown in 4-in. pots in a glass-house. Female flowers were emasculated the day before anthesis and pollen from the male flowers transferred to them the same day.

(iii) *Clones of F₁ and F₂ Plants*.—In order to identify the number of genes present in the resistant varieties, and the T.S.W. strains they controlled, it was desirable to inoculate each of the F₁ and F₂ plants with the 10 strains isolated earlier in this project.

When a number of virus strains are inoculated into a single plant, the interaction and masking effects produced make it impossible to assess the resistance or susceptibility of that plant to any one of the strains.

This problem was overcome by removing 10 shoots from each of the F_1 and F_2 plants and rooting them as separate clones. Each of these 10 genetically identical clones could then be inoculated with a different virus strain. This method was employed throughout this project as a practical means of obtaining the reaction of individual plants to a number of virus strains.

(b) Cultural Techniques

(i) *Clone Production.*—The F_1 and F_2 plants were grown to a height of about 6 in. in 4-in. pots. The young growing tips were removed to encourage growth of laterals. The growing tips of the laterals were also removed. When there were 10-12 laterals approximately 3 in. long, they were removed and placed in seed boxes of damp sand. The cuttings from each plant were placed in separate rows suitably identified.

The cuttings were kept well watered for 2 weeks, by which time they had rooted and were then transferred into 3-in. pots. The pots were labelled with the original plant number, and a further number from 1 to 10 to facilitate the identification of any particular clone. The whole process from seeds to potted clones was done in an insect-proof glass-house.

(ii) *Experimental Procedures.*—When ready for inoculation, the 10-12 in. clones were transferred to a basement room which was maintained at a constant temperature of 85°F. and illuminated by two banks of fluorescent lights, giving a light intensity of approximately 500 ft.-candles.

The standardization of the inoculum, inoculating technique, and environmental conditions were identical to those described previously (Finlay 1952).

After inoculation all plants were maintained under the fluorescent lights for a period of about 40 days, and were then transferred to an insect-proof glass-house until some fruit had matured in order to check for delayed systemic infection.

The clones of each F_1 or F_2 population to be tested were arranged in a random manner, and inoculated in such a way that every 10 genetically identical clones were inoculated with the 10 virus strains, a different strain for each clone. Each F_1 population was represented by 30 plants. F_2 population size approximated 200 plants in each case. These numbers were multiplied by 10 by the use of 10 clones of each of the original F_1 or F_2 hybrid progeny. 10 parent plants were used in each of the tests for synergistic action between strains of T.S.W.

F_2 segregations were examined by the χ^2 test for goodness to fit to the expected Mendelian ratios.

III. RESULTS

(a) Identification of Genes Controlling Resistance to T.S.W. Virus in Tomatoes

The F_1 hybrids produced by crossing the four resistant tomato types with the susceptible variety Potentate exhibited three levels of resistance to various

strains of T.S.W. (Table 1). Some of the hybrids were resistant, others completely susceptible, and the remainder were susceptible, but delayed systemic infection was a noticeable feature with this class. Although these plants must be classed as susceptible, their resistance to the strains listed is certainly higher than those classed as susceptible. The plants of the control variety Potentate were fully susceptible to all strains.

It will be noted that there is a tendency for resistance to certain strains of the virus to be always associated with other strains, e.g. strains of TB₃, N₁, and R₁ are always found together in the same resistance group. This feature became fully apparent in the F₂ segregations where it was noted that the F₂ plants were resistant to groups of strains. There were five of these strain groups, and each F₂ plant which was resistant to one strain of a group was always resistant to the other strains of the same group. The groups of strains to which the F₂ plants were resistant are shown for each cross in Table 2.

TABLE 1
RESISTANCE IN THE F₁ OF CROSSES BETWEEN SUSCEPTIBLE AND T.S.W.-RESISTANT PARENTS TO 10 STRAINS OF T.S.W. VIRUS

Cross	No. of Plants Inoculated	T.S.W. Strains to Which F ₁ Plants are Resistant		
		Resistant	Susceptible—Delayed Systemic Infection	Susceptible
<i>L.p.</i> × S	30	TB ₃ , N ₁ , R ₁	TB ₁ , N ₂ , R ₂ , R ₃ , M ₁ , M ₂	TB ₂
T × S	30	TB ₂	TB ₁ , N ₂ , R ₂ , R ₃ , M ₁ , M ₂	TB ₃ , N ₁ , R ₁
PH × S	30	TB ₃ , N ₁ , R ₁	M ₁ , M ₂	TB ₁ , TB ₂ , N ₂ , R ₂ , R ₃
M × S	30	TB ₂	R ₂ , R ₃ , M ₂	TB ₁ , TB ₃ , N ₁ , N ₂ , R ₁ , M ₁
S × S	30	—	—	All 10 strains

L.p. = *L. pimpinellifolium*, T = Rey de los Tempanos, PH = Pearl Harbour, M = Manzana, S = susceptible variety Potentate.

The observed segregations were compared with the expected values by means of the χ^2 test. Monogenic inheritance is indicated for T.S.W. resistance to each of the strain groups.

From this evidence it appears logical to assume that five single genes are responsible for plant resistance to each of these strain groups. The resistance gene controlling strains TB₃, N₁, and R₁ is apparently a single dominant, as also is the gene controlling strain TB₂. The rest of the strains come under the control of three recessive genes.

It is suggested that these five genes be given the symbols SW₁^a, SW₁^b, sw₂, sw₃, and sw₄ as shown in Table 3. This table also summarizes the F₁ and F₂ behaviour of these genes. The plant reaction to virus strains M₁ and M₂ is one of immunity. Plants possessing genes SW₁^a, SW₁^b, sw₂, and sw₃ were resistant to the particular T.S.W. strains that these genes control, and gave localized infection following inoculation, but were later able to continue growth free of the disease.

The number of genes for T.S.W. resistance possessed by the five tomato types used in this study varied from none in the fully susceptible variety Potentate to four in both *L. pimpinellifolium* and Rey de los Tempranos (Table 4). No one of these tomato types carried all five genes.

(b) Tests for Allelism

To discover which, if any, of the five resistance genes were allelic, crosses were made between all the resistant tomato types in all possible combinations and the F₂ populations were tested with the 10 T.S.W. strains. The results are recorded in Table 5.

TABLE 2
NUMBERS OF RESISTANT AND SUSCEPTIBLE PLANTS IN THE F₂ PROGENY FROM CROSSES BETWEEN SUSCEPTIBLE AND T.S.W.-RESISTANT PARENTS

Cross	Strains to Which Plants are Resistant	No. of Plants Inoculated	Observed No. of Resistant Plants	Expected No. of Resistant Plants	Deviation	χ^2	P
<i>L.p.</i> × S	TB ₃ , N ₁ , R ₁	244	188	183	5	0.520	0.30-0.50
	TB ₁ , N ₂ , R ₂ , R ₃	244	63	61	2	0.087	0.70-0.80
	R ₂ , R ₃ , M ₂	244	54	61	7	1.071	0.30-0.50
	M ₁ , M ₂	244	59	61	2	0.087	0.70-0.80
T × S	TB ₁ , N ₂ , R ₂ , R ₃	237	66	59.25	6.75	1.024	0.30-0.50
	R ₂ , R ₃ , M ₂	237	57	59.25	2.25	0.113	0.70-0.80
	M ₁ , M ₂	237	62	59.25	2.75	0.170	0.50-0.70
	TB ₂	237	179	177.75	1.25	0.035	0.80-0.90
PH × S	TB ₃ , N ₁ , R ₁	198	147	148.5	1.5	0.200	0.80-0.90
	M ₁ , M ₂	198	56	49.5	6.5	1.139	0.20-0.30
M × S	R ₂ , R ₃ , M ₂	193	41	48.25	7.25	1.453	0.20-0.30
	TB ₂	193	142	144.75	2.75	0.209	0.50-0.70

L. pimpinellifolium and Pearl Harbour possess the dominant gene SW₁^a controlling resistance to strains TB₃, N₁, and R₁, but they are susceptible to strain TB₂. Rey de los Tempranos and Manzana have the dominant gene SW₁^b, making them resistant to strain TB₂, but they are susceptible to strains TB₃, N₁, and R₁.

It will be seen from Table 5 that, in any cross which brings together these two dominant genes SW₁^a and SW₁^b, the F₂ populations segregate one plant resistant to strains TB₃, N₁, and R₁, but susceptible to TB₂, two plants resistant to TB₂, TB₃, N₁, and R₁ and one plant resistant to TB₂ but susceptible to strains TB₃, N₁, and R₁. No plants susceptible to all these strains are obtained. These results indicate that SW₁^a and SW₁^b are a single pair of alleles governing the reaction to the two groups of strains TB₃, N₁, and R₁; and TB₂. The χ^2 test shows a good fit to a 1:2:1 ratio in the F₂ in each case where alleles SW₁^a and SW₁^b are brought together by crossing.

The heterozygous plants, $SW_1^a SW_1^b$, are resistant to both groups of strains. Under a field epidemic of T.S.W. in which the strains TB_2 , TB_3 , N_1 , and R_1 are present, $SW_1^a SW_1^b$ plants would prove more resistant than either SW_1^a or SW_1^b plants. The recessive genes sw_2 , sw_3 , and sw_4 appear to be inherited quite independently.

TABLE 3
INDEX OF GENE SYMBOLS FOR T.S.W. RESISTANCE IN TOMATOES

Gene Symbol	Resistance to T.S.W. Strains	Behaviour in F_1	F_2 Segregation		
			Immune	Resistant	Susceptible
SW_1^{a*}	TB_3, N_1, R_1	Resistant		3	1
SW_1^b	TB_2	Resistant		3	1
sw_2	TB_1, N_2, R_2, R_3	Susceptible†		1	3
sw_3	R_2, R_3, M_2	Susceptible†		1	3
sw_4	M_1, M_2	Susceptible†	1		3

* Capital letters refer to dominant genes.

† Susceptible but with delayed systemic infection.

The results recorded in Table 5 show the chances of obtaining useful resistance from a breeding programme incorporating any two of the resistant varieties used. The cross between *L. pimpinellifolium* and Rey de los Tempranos produces 50 per cent. of the F_2 plants resistant to all 10 strains of the virus. Only plants having the two dominant alleles SW_1^a , SW_1^b are completely

TABLE 4
T.S.W. RESISTANCE GENES PRESENT IN FOUR T.S.W.-RESISTANT TOMATO TYPES

Tomato Type	Genes Present
<i>L. pimpinellifolium</i>	SW_1^a, sw_2, sw_3, sw_4
Rey de los Tempranos	SW_1^b, sw_2, sw_3, sw_4
Pearl Harbour	SW_1^a, sw_4
Manzana	SW_1^b, sw_3

resistant, and, therefore, it is impossible to produce homozygous T.S.W.-resistant plants using these two parents. This also applies to *L. pimpinellifolium* crossed with Manzana, and Rey de los Tempranos crossed with Pearl Harbour. All the other crosses lack one of the genes governing resistance to some of the T.S.W. strains.

It is apparent from these results that it is impossible to breed a homozygous tomato variety completely resistant to all known strains of T.S.W. by using these four resistant tomato types as parents.

TABLE 5
NUMBERS OF RESISTANT AND SUSCEPTIBLE PLANTS IN THE F₂ PROGENY BETWEEN T.S.W.-RESISTANT PARENTS

Cross	Genes	F ₂ Segregations			Observed No. of Resistant Plants	Expected No. of Resistant Plants	Deviation	χ ²	P
		Im- mune	Resis- tant	Sus- ceptible					
L.p. × T (223)†	SW ₁ ^a SW ₁ ^b	1 : 2 : 1*			49	55.75	6.75	1.404	0.30-0.50
				109	111.50	2.50			
				65	55.75	9.25			
	sw ₂	All resistant			—	—	—		
	sw ₃	All resistant			—	—	—		
	sw ₄	All resistant			—	—	—		
L.p. × PH (263)	SW ₁ ^a	All resistant			—	—	—		
	sw ₂	1	3		60	65.75	5.75	0.671	0.30-0.50
	sw ₃	1	3		62	65.75	3.75	0.285	0.50-0.70
	sw ₄	All immune			—	—	—		
L.p. × M (196)	SW ₁ ^a SW ₁ ^b	1 : 2 : 1			47	49	2	1.470	0.30-0.50
				106	98	8			
				43	49	6			
	sw ₂	1	3		43	49	6		
	sw ₃	All resistant			—	—	—		
	sw ₄	1	3		45	49	4	0.435	0.50-0.70
T × PH (217)	SW ₁ ^a SW ₁ ^b	1 : 2 : 1			55	54.25	0.75	0.705	0.70-0.80
				103	108.50	5.50			
				59	54.25	4.75			
	sw ₂	1	3		60	54.25	5.75		
	sw ₃	1	3		64	54.25	9.75	2.336	0.10-0.20
	sw ₄	All immune			—	—	—		
T × M (207)	SW ₁ ^b	All resistant			—	—	—		
	sw ₂	1	3		49	51.75	2.75	0.195	0.50-0.70
	sw ₃	All resistant			—	—	—		
	sw ₄	1	3		56	51.75	4.25	0.465	0.30-0.50
PH × M (187)	SW ₁ ^a SW ₁ ^b	1 : 2 : 1			51	46.75	4.25	1.428	0.30-0.50
				96	93.50	2.50			
				40	46.75	6.75			
	sw ₃	1	3		39	46.75	7.75		
	sw ₄	1	3		46	46.75	0.75	0.016	0.80-0.90

* 1:2:1 Segregation for resistance = 1 resistant (R) to SW₁^a but susceptible (S) to SW₁^b : 2 R to SW₁^a and SW₁^b : 1 R to SW₁^b but S to SW₁^a.

† The number of F₂ plants used in determining the segregations for resistance in each of the crosses.

(c) Testing for Synergism of T.S.W. Strains

Some mild and ringspot strains of T.S.W., when associated with a severe strain of the virus, were able to gain access to, and multiply in, host plants which were normally resistant to them in their pure form. This has been cited by Norris (1951) and Finlay (1952) as evidence of synergism.

The partially resistant tomato plants were inoculated with a strain of the virus to which they were susceptible. When infection had become systemic, usually after about 2 weeks, the plants were inoculated with different strains of the virus to which the plants were normally resistant. The results are recorded in Table 6.

TABLE 6
SYNERGISTIC EFFECTS OF T.S.W. STRAINS

Tomato Type	Initial Inoculation*	Plant Reaction to Strains to Which They are Normally Resistant	
		Susceptible	Resistant
Potentate	M ₂	TB ₂ , N ₁ , R ₂	—
<i>L. pimpinellifolium</i>	TB ₂	TB ₃ , N ₁ , R ₂ , M ₁	—
Pearl Harbour ..	R ₂	N ₁	TB ₃
	R ₂ +N ₁	TB ₃	—
	N ₂	TB ₃	—
Manzana	M ₁	R ₂	TB ₂
	M ₁ +R ₂	TB ₂	—

* The tomato types are fully susceptible to these strains.

Potentate, a susceptible variety, was susceptible to all strains tested. Porter's strain of *L. pimpinellifolium*, infected with strain TB₂, allowed entry and multiplication of a full range of milder strains. It will be seen in Pearl Harbour, which was systemically infected with strain R₂, that infection with a Necrotic strain was necessary before TB₃ could successfully multiply. A similar result was recorded for Manzana infected with M₁, but infection with R₂ did allow entry of strain TB₂.

The mechanism of this reduction in the plant's resistance is not known. The virus-inactivating system in the plant may be inhibited by infection with a tip blight or Necrotic strain, or the by-products of infection from a severe strain supply substances necessary for the successful multiplication of the milder strains which are normally unable to exist in this resistant host plant in pure form. Initial infection with a mild strain allows entry and multiplication of strains in an increasing order of their severity, thus producing a gradual breakdown of the inactivating system over a period of some weeks.

These results substantiate the earlier evidence that a synergism of T.S.W. strains exists in their host plants.

IV. DISCUSSION

During the past three decades, all the breeding programmes designed to incorporate T.S.W. resistance into commercially acceptable tomato varieties have been carried on without adequate knowledge of the inheritance of resistance in T.S.W.-resistant parents. The lack of a satisfactory method of selecting resistant or susceptible phenotypes in hybrid populations is suggested as being the basic cause for this lack of knowledge.

Kikuta, Hendrix, and Frazier (1945) were able to identify a single dominant gene controlling T.S.W. resistance in Pearl Harbour. It is likely that the gene was SW_1 , controlling resistance to strains TB_1 , N_1 , and R_1 .

Holmes (1948) indicated the presence of a single recessive gene in Rey de los Tempranos as being responsible for its T.S.W. resistance in New Jersey, where the normal field complex of the virus produced tip blight symptoms on susceptible tomatoes and ringspot symptoms on dahlias. The gene was probably sw_2 , controlling strains TB_1 , N_2 , R_2 , and R_3 .

These varieties grown in the localities mentioned would not have given a monogenic segregation had there been any other strains of the virus present at the time. If other strains had been present in the local complex these varieties would have either been susceptible, or the added strains would have been controlled by another gene or genes.

From the results recorded in this paper it will be seen that when Pearl Harbour was grown in New Jersey, the major gene necessary to control T.S.W. infection in that area would be absent, thus accounting for the susceptibility of this variety in a different locality.

Workers such as Hutton and Peak (1949) in Canberra, and Smith and Gardner (1951) in California, used Porter's strain of *L. pimpinellifolium* as a source of T.S.W. resistance, in areas where a large number of T.S.W. strains were present in the field complex. They were unable to obtain evidence of simple Mendelian inheritance of resistance.

Hutton and Peak (1952) suggest that the inoculation of hybrid plants at a temperature of 90°F. with the Ringspot strain will facilitate the selection of resistant or susceptible phenotypes. The results contained in this paper indicate that it would be necessary to identify which ringspot strain or strains was being used. The use of ringspot strains would give no indication of the presence or absence of genes sw_4 and SW_1^b . If these genes were absent in the selected phenotypes there could be a disorganization of the plant's virus-inactivating system by a mild strain due to synergistic action.

As there are five genes for resistance to T.S.W., and each gene controls resistance to a group of strains, it is suggested that at least one strain from each group be used in inoculation of clones of hybrid progenies to facilitate the selection of resistant and susceptible phenotypes. Further, it is suggested that the standardized environmental conditions described previously (Finlay 1952) be used, because the results obtained were dependent on this mode of testing.

It is now possible to assess the relative usefulness of these four resistant tomato types as parents in a breeding programme designed to develop spotted-wilt-resistant tomatoes with desirable agronomic characters.

Porter's strain of *L. pimpinellifolium* possesses resistance to all known T.S.W. strains except TB₂, but one or more of the resistance genes are apparently linked with small fruit size and certain other characters of this species.

Hutton and Peak (1952) suggest that Rey de los Tempranos is a better source of resistance than *L. pimpinellifolium* because it has a more efficient inactivating system, has larger fruit, and crosses very readily with commercial varieties. As Rey de los Tempranos carries the same resistance as *L. pimpinellifolium*, except that it has the allele SW₁^b instead of SW₁^a, the suggestion appears to be a useful one.

The occurrence of strain TB₂, which is controlled by allele SW₁^b, has only been recorded in Western Australia (Finlay 1952). For other areas it may be sufficient to replace SW₁^b in Rey de los Tempranos by its allele SW₁^a from Pearl Harbour, and then breed for fruit size and other desirable agronomic characteristics.

Pearl Harbour appears to be useful as a source of the SW₁^a allele, much better than *L. pimpinellifolium* because of its large, high-quality fruit.

Manzana, apart from its large fruit, has little to recommend it as a parent when breeding for T.S.W. resistance, because the two resistance genes it possesses are also to be found as part of the gene complement in Rey de los Tempranos.

Of the four tomato types used in this study, the combination of genes from the varieties Rey de los Tempranos and Pearl Harbour appear to offer the best approach in breeding for T.S.W. resistance, either by the replacement of the SW₁^b allele in Rey de los Tempranos by SW₁^a from Pearl Harbour and breeding a variety homozygous for resistance, or by using the F₁ hybrid produced by crossing these two varieties. If the F₁ hybrid were to be used it would be necessary to increase the fruit size of Rey de los Tempranos before crossing with Pearl Harbour. The delay of systemic infection caused by the action of the heterozygotes of genes sw₂ and sw₃ is probably sufficient to allow a normal crop of fruit to be produced free from symptoms, as reported by Finlay (1951). Increased yield of up to 50 per cent. due to hybrid vigour (Finlay 1951) adds to the benefits to be gained by using the F₁ hybrid of these two varieties.

Complete immunity to all strains of spotted wilt virus could not be obtained from any combination of the four tomato types used in these experiments. Because of its immunity to T.S.W. under field conditions, *L. peruvianum* appears to warrant further study as a parent for use in producing tomato varieties to combat the ever-increasing menace of the spotted wilt virus.

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