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LEAF SHRIVELLING VIRUS DISEASES OF THE TOMATO.

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SUMMARY.

Three virus diseases of the tomato recorded in south-eastern Queensland are described. They are tomato leaf shrivel, tomato yellow shrivel and tomato fern-leaf shrivel.

From the late autumn to the spring of each year, tomato leaf shrivel is widespread in south-eastern Queensland, exhibiting total infection in many crops. Diseased plants have an etiolated and unthrifty appearance, with foliar symptoms influenced by the period after plant infection, the prevailing temperatures and, to a lesser extent, the variety of tomato. During winter, advanced symptoms consist of a diffuse light-green (or yellow-green) and dark-green leaf mottle, a general leaflet rugosity and a downwards and inwards recurving of leaves and leaflets, which are reduced in size. Recently expanded leaves develop a variable marginal leaf necrosis and an interveinal and veinal necrosis on the under-surface. Shrivelling of the lower leaves is characteristic and advances systematically upwards.

The virus responsible for the disease is transmitted by mechanical inoculations, by three aphid species (in order of vector efficiency, Myzus persicae Sulz., Macrosiphum solanifolii Ash. and Aphis gossypii Glov.), and by grafting. The characteristics of the virus established that it is a leaf shrivelling strain of potato virus Y.

The rapid field spread of the tomato leaf shrivel is due to infective aphid vectors; mechanical transference during cultural operations contributes to a minor degree.

Three alternative weed hosts of the virus are Solanum nigrum L., Nicandra physaloides Gaertn. and Physalis peruviana L.

The disease may be responsible for substantial reductions in tomato fruit yields. Yield losses are modified by the stage of plant development at the time of infection and the prevailing temperatures during fruit development.

The assessment of tomato varieties with respect to their virus susceptibility is described.

Tomato yellow shrivel disease is caused by a virus complex which has been resolved into two components—the leaf shrivelling strain of potato virus Y and the aucuba strain of tobacco mosaic virus. The variable expression of the disease is typified by a bright yellowing of leaves, interveinal puckering, recurving downwards and inwards of leaves and leaflets and an extensive shrivelling of the lower leaves. Diseased plants develop an internal vascular and pith necrosis of the stem tissues which may eventually lead to a stem collapse and wilting. Fruit-setting ceases on infection, and ripening fruit may exhibit yellowish blotches. Fruit production is seriously impaired by the disease.

Tomato fern-leaf shrivel disease, which is restricted to the late winter and early spring in the districts surrounding Brisbane, is caused by a further virus complex composed of the leaf shrivelling strain of potato virus Y and cucumber mosaic virus.

I. INTRODUCTION.

During the past decade, three closely related diseases of the tomato which do not conform in symptoms to any one of the many defined diseases appeared regularly each year in commercial plantings in the horticultural districts of south-eastern Queensland. Recently two of these diseases were observed in other coastal districts of Queensland.

During 1953-54, studies in the glasshouse and in the field established that the diseases were of virus origin. A leaf shrivelling strain of *potato virus Y* was isolated from tomato plants expressing symptoms of a tomato leaf shrivelling disease. Two other virus diseases, referred to as tomato yellow shrivel and tomato fern-leaf shrivel, were found to be caused by virus complexes with the leaf shrivelling strain of *potato virus Y* as a common virus component. These three diseases are dealt with separately in the following pages.

II. TOMATO LEAF SHRIVEL DISEASE.

Tomato production in Queensland has been seriously affected by a hitherto unrecorded virus disease which is known locally as tomato leaf shrivel. The name of the disease refers to one of its prominent symptoms—shrivelling of the basal leaves.

A sudden decline in the winter production of trellised and cradled tomato crops from 1945 onwards probably marks the widespread appearance of the disease in south-eastern Queensland. The first known observation of a tomato disease which closely resembled that caused by tomato leaf shrivel was made by J. E. C. Aberdeen (personal communication) at Bowen in 1948.

Surveys of tomato plantings in the districts surrounding Brisbane during the period 1954–1956 established that tomato leaf shrivel was seriously affecting the production of all crops cultivated during the late autumn, winter and spring months of each year. A significant feature of the disease in these districts was its rapid spread—plantings became totally infected at or prior to the early fruit harvest (Fig. 1).

In the warm coastal areas of Queensland the disease was also located in winter tomato plantings within the horticultural districts of Nambour, Rockhampton and Bowen.

There is a single overseas record of a virus disease which is very similar to the tomato leaf shrivel disease. This disease, which is caused by *potato virus Y*, was observed by Conover and Fulton (1953) in tomato fields at Dade County, Florida.

Fig. 1.

A Planting of Tomatoes (Variety Q2) Totally Infected with Tomato Leaf Shrivel Disease.

(1) Symptoms.

Tomato leaf shrivel is easily overlooked during the early stages of symptom expression, as it tends to be obscured by plant uniformity within totally infected plantings.

The order of expression and the range of disease symptoms were ascertained by infecting experimental tomato plants, both in the field and in the glasshouse, with isolates of the virus. Variations in symptom expression were influenced or directly affected by the period after infection, the prevailing temperatures and, to a lesser extent, the variety of tomato. The typical symptoms of the variety Q2 in order of their appearance are summarised below.

Two weeks after infection.—Terminal growth exhibited a variable light-green and dark-green leaf mottle with yellowing of the basal portion of leaflets; interveinal puckering; and recurving downwards and inwards of leaves and leaflets. Fully expanded leaves were unchanged in appearance (Fig. 2).

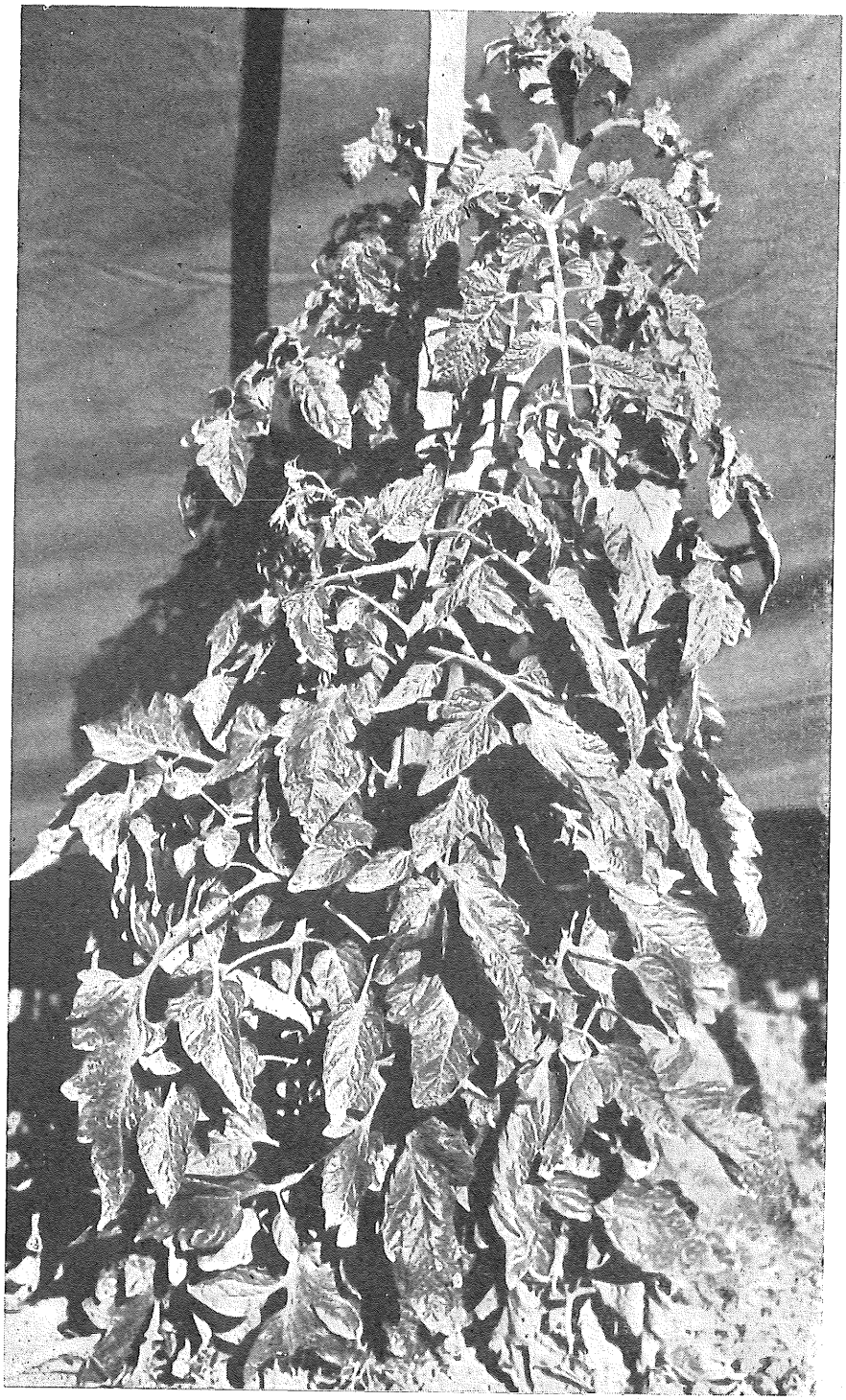

Fig. 2.

Symptoms Displayed by a Tomato Plant (Variety Q2) Inoculated Two Weeks Previously with *P.V.Y.l.s.*

Three weeks after infection.—General light-green, yellow-green and dark-green mottling of the terminal leaves; obscure green vein banding; general leaflet rugosity; and slight reduction in size of leaves were shown. Terminal and fully expanded leaves, which were of harsh appearance and brittle texture, were recurved downwards and inwards. Recently expanded leaves developed traces (or areas) of interveinal and veinal necrosis on the under-surfaces of leaflets, together with a variable marginal and spot necrosis (Fig. 3).

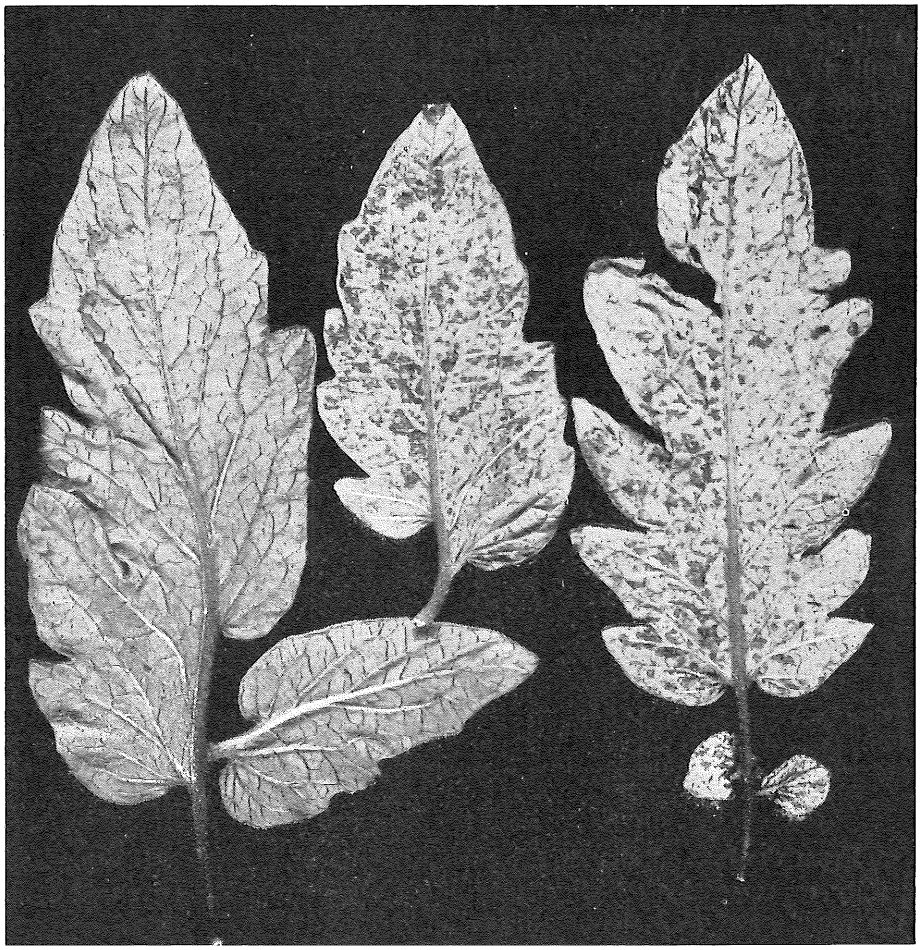

Fig. 3.

Interveinal and Veinal Necrosis on Under-surface of Leaflets Appearing Three Weeks After Infection With *P.V.Y.l.s.* Healthy leaflet on left.

Four to six weeks after infection.—The marginal necrosis of leaflets extended inwards towards the mid-veins of fully expanded leaves and the basal leaves were shrivelled and attached to the stem.

Four generalisations were derived by correlating symptoms exhibited by experimentally and naturally infected plants with mean monthly temperatures. The mean monthly temperatures for 1954 and 1955 for the glasshouse and for the Brisbane Meteorological Bureau about a mile distant are listed in Table 1.

When mean temperatures were less than 60 deg. F., symptoms were sharply defined and accentuated by the presence of necrosis. From 65 deg. to 70 deg. F. the necrotic phases of the disease were present only in trace amounts or entirely absent. From 70 deg. to 75 deg. F., symptoms were mild and difficult to detect. With mean temperatures greater than 75 deg. F. the symptoms were masked.

Table 1.

MEAN MONTHLY TEMPERATURES FOR 1954–55, BRISBANE.

Month.	Mean Temperature (°F.).			
	1954.		1955.	
	Observatory.	Glasshouse.	Observatory.	Glasshouse.
January	74·5	85·2	76·6	87·9
February	76·4	83·1	78·0	84·7
March	76·8	85·9	75·3	80·0
April	70·8	77·9	71·8	79·5
May	64·5	70·9	64·6	72·7
June	60·3	67·2	60·6	71·6
July	61·1	66·9	58·3	68·8
August	60·9	69·7	61·3	73·9
September	63·6	74·4	65·0	76·9
October	68·1	74·7	71·2	81·0
November	73·1	79·7	73·1	84·8
December	74·7	86·1	75·4	82·7

From the late autumn to the spring months of each year the symptoms generally expressed by tomato plants of all varieties in the advanced stages of the tomato leaf shrivel disease were observed to be as follows:—

A mild yellow-green or light-green and dark-green diffuse leaf mottle; interveinal puckering of leaflets appearing as a general leaf rugosity; downwards and inwards recurving of leaves and leaflets; varying degrees of marginal leaf necrosis and necrotic spots or areas in fully expanded leaves; shrivelling of the basal leaves progressing slowly upwards; reduction in size of leaves, leaflets and thickness of stems; reduction in fruit numbers; and slight reduction in fruit size (Fig. 4).

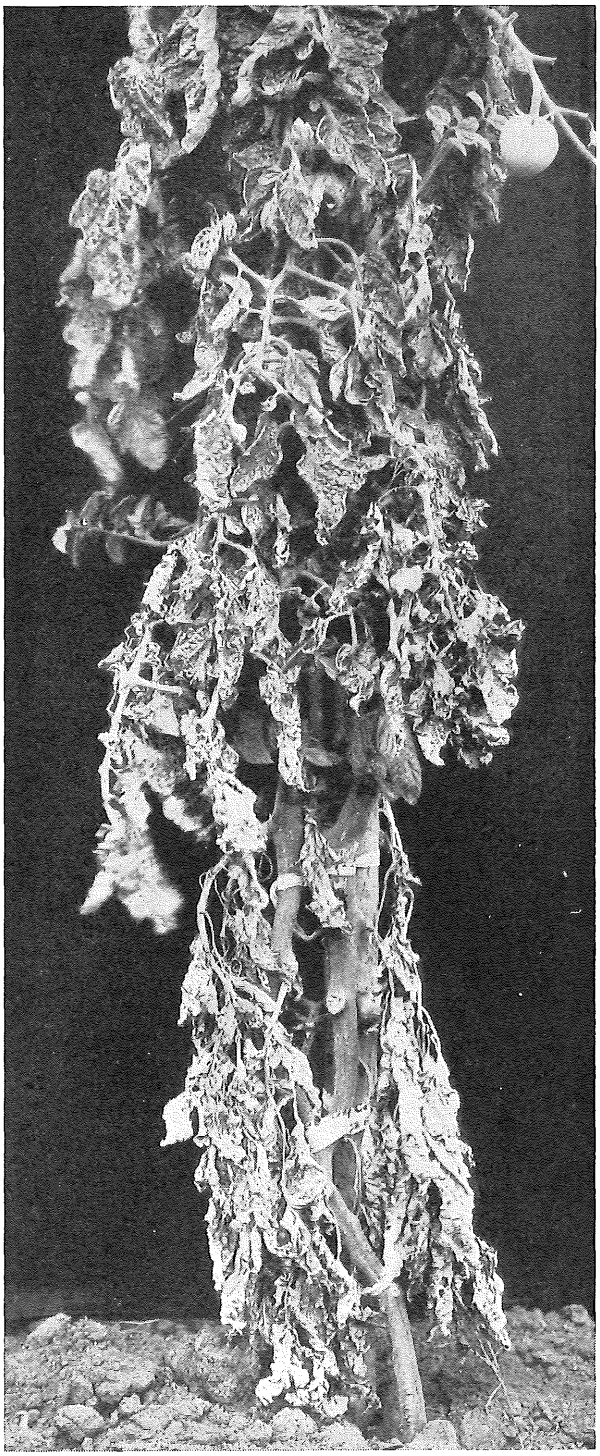

Fig. 4.

Advanced Leaf Shrivelling Symptoms Shown by a Tomato Plant (Variety Q2) in the Late Stages of Infection with *P.V.Y.l.s.*

The high temperatures during the summer months were found to mask symptoms. With increasing day temperatures from late spring to early summer, less necrosis developed in the expanding leaves. As growth of affected plants continued during the summer months, terminal leaves of plants appeared to be free of the disease. All traces of infection soon disappeared in young plantings which had shown early symptoms.

More than 50 diseased varieties and unnamed selections were observed in field plantings during the winter and spring months. The variations in disease expression within plants of different varieties were quantitative rather than qualitative. One variety of German origin exhibited more acute symptoms of the disease than any other diseased variety (Fig. 9).

During the winter months, a feature of the disease was the appearance of an interveinal and veinal necrosis on the under-surface of recently expanded leaves approximately three weeks after plant infection. On a few subsequently expanded leaves, diminishing areas of interveinal necrosis were formed and the symptom eventually did not appear in young expanding leaves (Fig. 3). This symptom was considered to be either a shock reaction or a plant response to infection during prevailing low temperatures. During periods of high wind velocities, brittle leaflets were found to tear along lines of necrosis extending from leaf margins to the mid-veins, giving the lower regions of diseased plants a ragged appearance.

Another feature of the disease was the etiolated appearance of diseased plants. This effect was most prominent in plants which were staked or otherwise supported. It was due to the substantial reduction in the size of recurved leaves and leaflets and the thickness of plant stems, unaccompanied by any measurable reduction in the distance between leaf nodes.

Fruit set following infection was seriously impaired and mature fruit from diseased plants were smaller than fruit from healthy plants. The total yield of fruit from diseased plants was governed by the stage of plant development at the time of infection (Fig. 5).

Experimental tomato crops, cultivated under glass during the winter, exhibited mild symptoms of tomato leaf shrivel disease. The various necrotic phases were absent or appeared to a lesser degree. The mild disease expression was ascribed to the warm conditions in the glasshouse.

(2) Etiology of the Disease.

Over the past decade many of the tomato irregularities which were due to tomato leaf shrivel disease have been accepted as winter growth variations. The more obvious symptoms were variously ascribed to nutritional deficiencies, the phytotoxic effects of fungicides or insecticides or even fungal leaf infections.

During the autumn of 1954 diseased plants were collected from totally infected plantings to determine if a virus infection was responsible for the disease. The inoculation of a replicated range of test plants with sap extracts from diseased plants established the presence of a virus within diseased material. A mild expression of the field disease was reproduced in the glasshouse by inoculating tomato seedlings with expressed sap from virus-infected test seedlings.

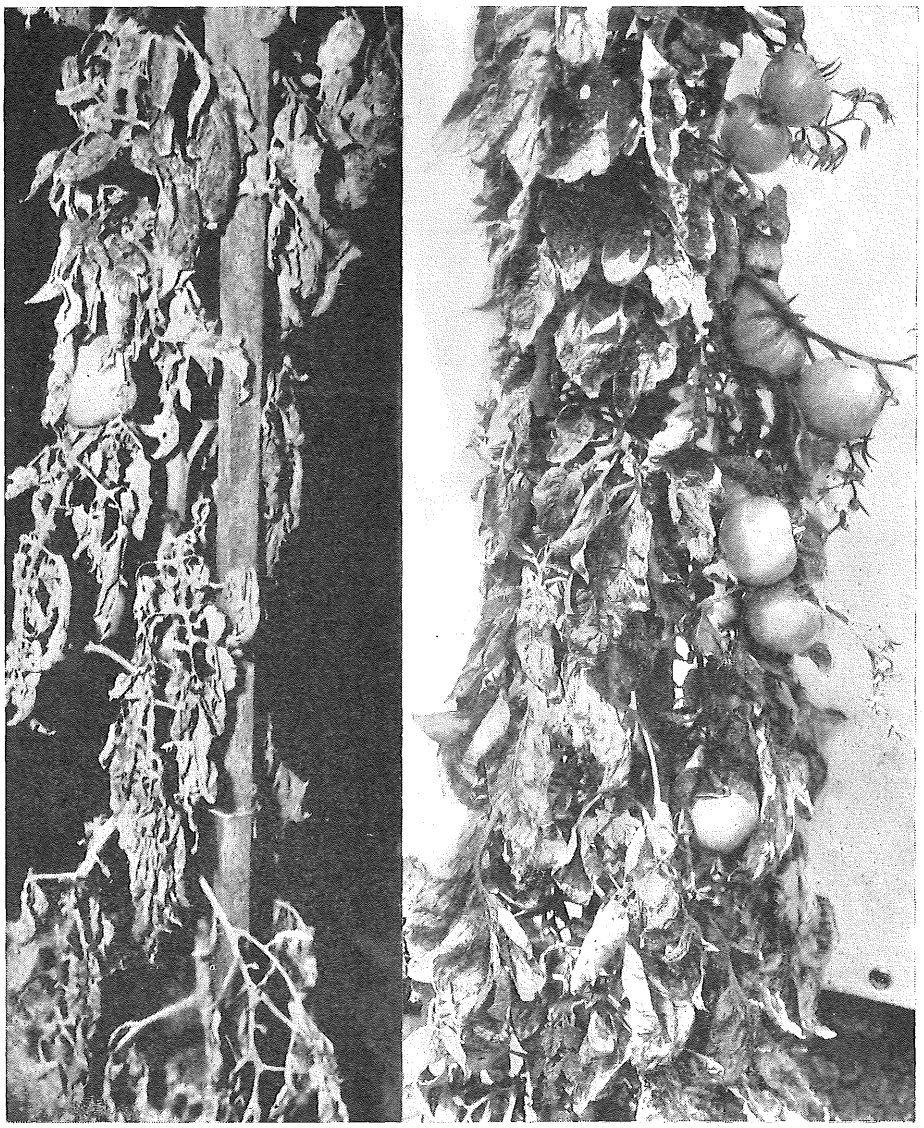

Fig. 5.

Tomato Leaf Shrivel Glasshouse Yield Trial. Left, plant inoculated at first flowering with *P.V.Y.l.s.* Right, healthy plant at same stage of development.

(3) Characteristics of the Virus.

Studies of the virus characteristics, which included methods of virus transmission, host range and physical properties, were undertaken in an insect-proofed glasshouse to identify the virus responsible for tomato leaf shrivel disease.

(a) Methods of Transmission.

(i) *Mechanical Inoculation.*—The virus was readily transmitted to susceptible hosts by mechanical inoculations.

Materials and methods.—Field isolates of the virus were maintained in young stock plants (*Nicotiana glutinosa* L. and *Nicotiana tabacum* L. var. White Burley) by regular sap transfer at intervals of 3–4 weeks. Paired seedlings were raised in clay pots containing steam-sterilized soil. One seedling was subjected to the virus treatment and the second seedling was retained as a check.

The following standard technique was used for studies involving mechanical inoculations. The terminal growth of virus-infected plants was macerated in a mortar, using a phosphate buffer solution and reducing agent (Norris 1946) as the extracting medium; extracted plant sap was expressed through a layer of marquisette and approximately 1 per cent. (w./v.) of Hyflo Super-Cel (Hutton and Peak 1951) was added to the extract as a leaf abrasive; infected sap was transferred to the leaves of test plants by lightly rubbing the sap across the upper leaf surfaces with a glass rod which was flattened and bent at one end through an angle of 75 deg.; virus inoculum was washed from treated leaves; potted seedlings were inoculated at least in quintuplicate.

During the cool months of the year transmission was achieved in most experiments by transferring the virus mechanically from stock plants to other susceptible plants. The mechanical inoculation technique was employed to determine the host range and physical properties of the virus.

(ii.) *Insect Transmission.*—Three aphid species—*Myzus persicae* Sulz., *Macrosiphum solanifolii* Ash. and *Aphis gossypii* Glov.—were found to transmit the virus. Details of the experiments are given in the section dealing with vector studies.

(iii.) *Graft Transmission.*—Three grafting techniques were used to transmit the virus from diseased to healthy plants. Potato scions were cleft-grafted and irrigated-approach-grafted to tomato stocks; tomato scions were cleft-grafted to potato stocks; potato and tomato plants were inarch-grafted. There were 74 successful unions. The tomato portion of each union was inoculated mechanically with the virus, which was transmitted in each instance through the graft-union to the potato portion. The virus was recovered from the potato by routine tests.

(b) Host Range.

Twenty-one species belonging to four botanical families—*Solanaceae* (14 species), *Leguminosae* (3 species), *Cucurbitaceae* (3 species) and *Chenopodiaceae* (1 species)—were mechanically inoculated with field isolates of the virus. The following 12 solanaceous species were susceptible to the virus:—

Nicotiana tabacum
Nicotiana glutinosa
Nicotiana sylvestris Speg. and Com.
Physalis floridana Rydb.
Physalis peruviana L.
Nicandra physaloides Gaertn.
Capsicum frutescens L.
Lycopersicon esculentum Mill.
Lycopersicon peruvianum Mill.
Lycopersicon pimpinellifolium Mill.
Solanum nigrum L.
Solanum tuberosum L.

The following nine species were immune to infection by mechanical inoculation:—

Solanum melongena L. var. Black Beauty
Datura stramonium L.
Pisum sativum L. var. Greenfeast
Lupinus angustifolius L. var. New Zealand Blue
Vigna sinensis (L.) Endl. ex Hassk. var. Black-eye 5
Cucumis sativus L. var. Early Fortune
Cucurbita pepo L. var. Long Cream Running
Citrullus vulgaris Schrad. var. Hawkesbury Wilt Resistant
Beta vulgaris L.

Plants of all species were rechecked for the presence of the virus.

(c) Symptoms.

The expression of the virus by susceptible species may be summarised as follows. (The temperatures given are the mean daily temperatures calculated for a period of 28 days following plant inoculation.)

Nicotiana tabacum vars. White Burley, Smyrna (64 deg. F.).—

Systemic: terminal vein clearing, marginal edge wave; small scattered areas of dark-green tissue in yellowed leaves, green vein banding; shrivelling of basal leaves; growth stunting.

Nicotiana glutinosa (71 deg. F.).—

Systemic: terminal vein clearing with associated areas of chlorosis, interveinal puckering or ballooning; variable spot necrosis with traces of vein necrosis on the under-surfaces of lower leaves; yellowing and shrivelling of basal leaves; growth stunting.

Nicotiana sylvestris (73 deg. F.).—

Systemic: terminal light-green and dark-green diffuse leaf mottle; green vein banding, yellowing and shrivelling of basal leaves; variable necrotic stem lesion at the point of attachment of shrivelled leaves; growth stunting.

The stems of the three inoculated species of *Nicotiana* showed an internal yellow-brown vascular discoloration which progressed upwards from the stem base in accordance with the yellowing of the basal leaves. The leaf-yellowing symptom was followed by shrivelling and the shrivelled leaves remained attached to the stem.

Physalis floridana (75 deg. F.).—

Local: yellow and necrotic spots.

Systemic: terminal vein clearing, yellowing, interveinal puckering, downwards and inwards recurving of leaves followed by terminal vein necrosis; vascular necrosis of stem tissues; necrotic plant collapse (Fig. 6).

Physalis floridana (84 deg. F.).—

Systemic: terminal vein clearing, light-green and dark-green leaf mottle, mild interveinal puckering, downwards and inwards recurving of leaves; yellowing, variable leaf necrosis followed by shrivelling and abscission of basal leaves; growth stunting.

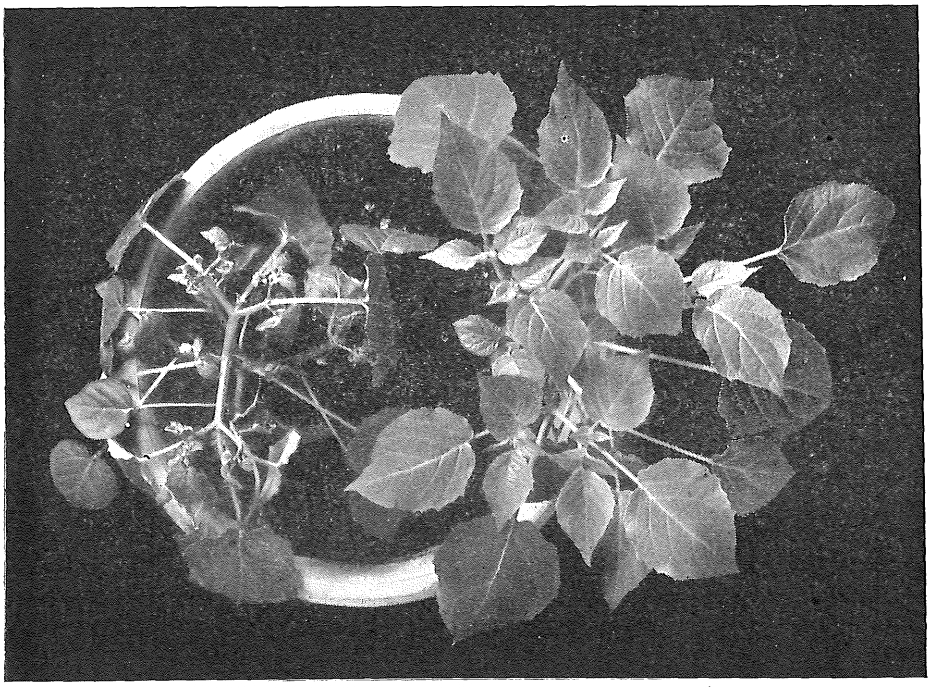

Fig. 6.

Susceptibility of *Physalis floridana*. Left, plant inoculated 11 days previously with *P.V.Y.l.s.* Right, healthy plant.

With increasing day temperatures in the glasshouse beyond 85 deg. F. the virus symptoms became masked. During the winter and early spring, *Physalis floridana* proved to be an excellent differential host for determining the presence of the virus in diseased material.

Physalis peruviana (69 deg. F.).—

Systemic: terminal vein yellowing, light-green and dark-green mosaic, interveinal puckering or ballooning; growth stunting.

Capsicum frutescens var. Floral Gem (76 deg. F.), *Nicandra physaloides* and *Solanum nigrum* (69 deg. F.).—

Systemic: terminal vein clearing, interveinal puckering, green vein banding; growth stunting.

Solanum tuberosum.—

The virus symptoms exhibited by plants of the potato variety Carman differed according to the method of virus transmission. The reaction in each case indicated virus hypersensitivity.

(*a*) *Mechanical inoculation* (69 deg. F.).—

Local: Necrotic lesions with associated traces of vein necrosis surrounded by yellow chlorotic areas.

Systemic: none. A similar local reaction was exhibited by plants of potato seedling 11–84 which were mechanically inoculated with the virus.

(*b*) *Tomato/potato graft transmission* (*i*) *var. Carman* (78 deg. F.).—

Systemic: terminal leaf chlorosis, variable interveinal puckering and necrotic spotting; numerous necrotic spots on expanding leaves; vein necrosis; coalescence of necrotic spots, leaf shrivelling; internal browning of stem; death of axillary side-shoots; necrotic collapse.

(*ii*) *Potato seedling* 11-79 (78 deg. F.).—

Systemic: terminal leaf chlorosis; upwards rolling of leaves; variable scattered necrotic areas; reduction in size of terminal leaves; growth stunting; systemic necrotic collapse.

When temperatures exceeded 80 deg. F., the potato portion of the grafts entered a recovery phase consisting of chlorotic lateral growth and scattered necrotic spots. Beyond 86 deg. F., Carman grafts developed free of virus symptoms.

Lycopersicon peruvianum and *L. pimpinellifolium* (73 deg. F.).—

Systemic: growth stunting only; virus recovered from inoculated plants.

Lycopersicon esculentum.—

As described previously.

(d) Physical Properties.

The four physical properties of the virus—thermal inactivation point, tolerance of the virus to dilution, longevity of the virus *in vitro*, and longevity of the virus in detached leaves—were studied.

(i.) *Thermal Inactivation Point.*—The technique used to determine the thermal inactivation point of the virus was a modified version of the method described by Best (1946). The heat-treated sap was mechanically inoculated into plants of *Nicotiana glutinosa.*

The thermal inactivation point ranged from 56 deg. C. to 60 deg. C. with different virus isolates. The experimental results are summarised in Table 2.

Table 2.

THERMAL INACTIVATION POINT.

Experiment Number	Results of Inoculating Test Plants with Heated Virus Extract.
1. (72°F.) ..	22°C.—8/8 ; 40°C.—6/6 ; 45°C.—6/6 ; 50°C.—6/6 ; 55°C.—3/6 ; 60°C.—0/6 ; 70°C.—0/6. (Temperature fluctuations during heating = ± 1°C.).
2. (82°F.) ..	25°C.—5/5 ; 54°C.—2/5 ; 56°C.—0/5 ; 58°C.—0/5 ; 60°C.—0/6. (Temperature fluctuations during heating = ± 0·5°C.)
3. (73°F.) ..	18°C.—5/5 ; 45°C.—3/5 ; 48°C.—5/5 ; 50°C.—4/5 ; 52°C.—2/5 ; 54°C.—1/5 ; 56°C.—1/5 ; 60°C.—0/5. (Temperature fluctuations during heating $\begin{cases} + 0{\cdot}5°C.) \\ - 0{\cdot}2°C.) \end{cases}$

(ii.) *Tolerance to Dilution.*—Raw undiluted sap was extracted from stock plants and added to measured quantities of buffer solution to give a range of dilutions. Test plants showed that the dilution end-points ranged from $1:5x10^2$ to $1:5x10^4$. Table 3 details the experimental range of dilutions which were used with three different virus isolates and the number of plant infections which were obtained with each dilution.

Table 3.

TOLERANCE TO DILUTION OF VIRUS-INFECTED SAP.

Dilution.	Plant Infections in 3 Experiments.		
	Expt. 1 (73°F.).	Expt. 2 (84°F.).	Expt. 3 (71°F.).
1	3/3	5/5	5/5
$1:10^2$	..	..	5/5
$1:5 \times 10^2$	..	..	0/5
$1:10^3$	1/3	..	0/5
$1:5 \times 10^3$	2/3	1/5	0/5
$1:10^4$	1/3	0/5	0/5
$1:5 \times 10^4$	0/3	0/5	0/5
$1:10^5$	0/3	0/5	..
$1:25 \times 10^4$	0/3	0/5	..
$1:5 \times 10^5$	0/3	..	..
$1:10^6$	0/3	..	..

(iii.) *Longevity of the Virus in vitro.*—Raw undiluted sap was retained in stoppered test tubes at a constant temperature of 27 deg C. At intervals of time sap was mechanically inoculated into plants of *Nicotiana glutinosa* in quintuplicate.

One virus isolate remained infectious in storage for 2 days but not longer than 6 days, and a second isolate remained infectious for 7 days but not longer than 9 days.

(iv.) *Longevity of the Virus in Detached Leaves.*—Leaves were removed from virus-infected plants and retained between blotting paper at room temperature (18–21 deg. C.). The virus remained infectious for 2 days and not more than 5 days in diseased tomato leaves. It remained infectious for not more than 8 days in the leaves of an infected plant of *Nicotiana glutinosa.*

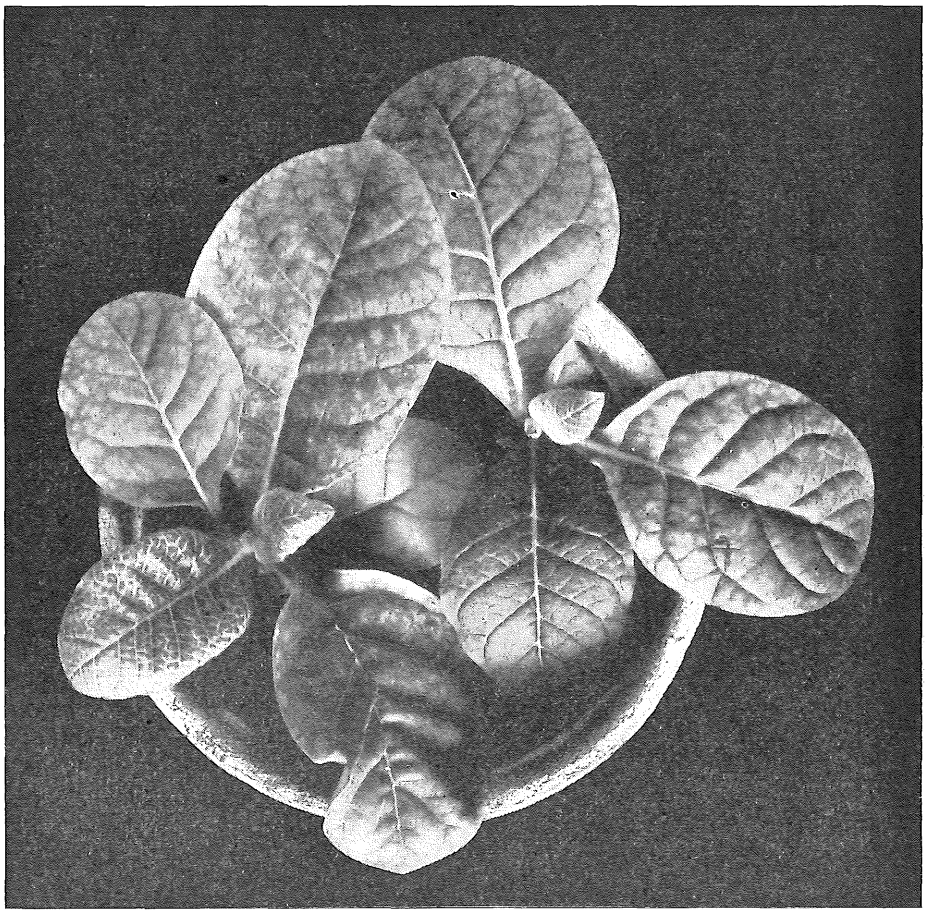

Fig. 7.

Interaction in Tobacco Plant. Left, symptoms produced on White Burley tobacco by *P.V.Y.l.s.* and *P.V.X.* Right, plant inoculated with *P.V.Y.l.s.* only.

(v.) *Interaction of the Virus and Potato Virus X in Tobacco Seedlings.*—Ten pairs of tobacco seedlings (var. White Burley) were mechanically inoculated, one seedling of each pair with *potato virus X* (*P.V.X.*), and, 24 hours later, all seedlings with the leaf shrivelling strain of *potato virus Y*. The tobacco seedlings inoculated with the two viruses exhibited severe vein yellowing, vein necrosis, variable necrotic spotting of leaves, interveinal puckering, and leaf yellowing followed by shrivelling of the basal leaves (Fig. 7).

(4) Identity.

The characteristics of the virus, together with the additional evidence presented in this section, suggest that the virus responsible for tomato leaf shrivel disease is a leaf shrivelling strain of *potato virus Y* (*P.V.Y.l.s.*). The similarities between the virus and the described or type strains of *potato virus Y* (*P.V.Y.t.*) are outlined below with reference to the characteristics of each strain.

The type and leaf shrivelling strains of *P.V.Y.* have the same methods of transmission. The type strains are readily transmitted by sap inoculations (Darby, Larsen and Walker 1951), by the aphid vectors *Myzus persicae* and *Macrosiphum solanifolii* (Bawden and Kassanis 1947), and by grafting (Smith 1931).

The host range and symptoms exhibited by solanaceous species inoculated with *P.V.Y.l.s.* are generally in accordance with those reported for *P.V.Y.t.* (Smith 1931, 1949; Koch 1933; Dennis 1938; Dykstra 1939; Ross 1948; Darby, Larsen and Walker 1951; Hutton and Peak 1952; Munro 1955). Notable variations from the *P.V.Y.t.* pattern of symptoms are the leaf shrivelling and necrotic phases of *P.V.Y.l.s.* expressed by the tomato, species of *Nicotiana* and *Physalis floridana*.

The thermal inactivation, tolerance to dilution and longevity *in vitro* end-points for *P.V.Y.l.s.* are within the normal range of variation as determined for *P.V.Y.t.* The wide range of physical properties reported for *P.V.Y.t.* (Koch 1933; Koch and Johnson 1935; Dykstra 1939; Darby, Larsen and Walker 1951; Sakimura 1953) include 52–62 deg. C. for the thermal activation point, $1:10^2$ to $1:75\times10^4$ for the dilution end-point, and from 1 day to 18 days for the longevity of *P.V.Y.t. in vitro*.

It is also shown that the mechanical inoculation of *P.V.X.* and *P.V.Y.l.s.* into tobacco seedlings, separately within 24 hours, produced the characteristic pattern of symptoms described by Bald and Pugsley (1941) and Darby, Larsen and Walker (1951) for *P.V.X.* and *P.V.Y.t.*

The effect of different temperatures on the symptoms expressed by a number of solanaceous species inoculated with *P.V.Y.l.s.* is similar to the temperature influence on the host—*P.V.Y.t.* reaction (Ross 1948; Darby, Larsen and Walker 1951; Hutton and Peak 1952).

A type (or potato) strain of *P.V.Y.* was obtained from R. D. Anderson, of Burnley, Victoria, for comparison studies with *P.V.Y.l.s.* Tomato plants (var. Q2) were cleft-grafted with potato scions of the variety Carman and potato selection 11–79 and infected with these two strains. Each strain induced a similar range of symptoms in the potato scions. There were quantitative differences in the necrotic phases of the two reactions; *P.V.Y.l.s.* was more severe than the *P.V.Y.t.* in Carman scions (11/11) and less severe in the 11–79 scions (5/5) (Fig. 8). At the same time, the two strains were mechanically inoculated into two separate groups of *Nicotiana glutinosa* and *Physalis floridana,* in quintuplicate. The main symptom differences were the mild systemic reaction by the *P.V.Y.t.* and the necrotic phases of the *P.V.Y.l.s.* reaction.

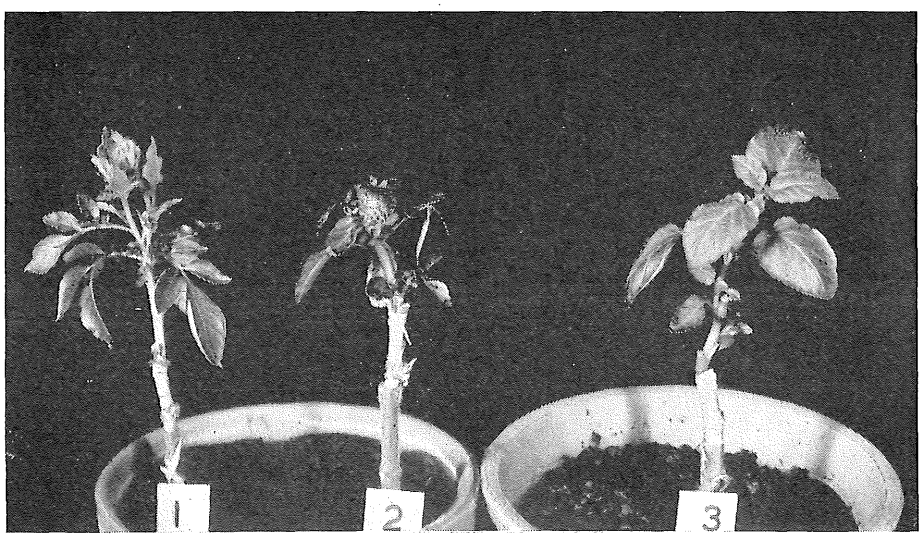

Fig. 8.

Potato Scions (Variety Carman) Grafted to Tomato Stocks (Variety Q2). 1, reaction due to *P.V.Y.t.* 2, reaction due to *P.V.Y.l.s.* 3, healthy plant.

The weight of evidence, discussed above, clearly demonstrates the identity of the virus responsible for the tomato leaf shrivel disease. In accordance with the system of virus nomenclature adopted by the Imperial Mycological Institute (1946) it is named *potato virus Y,* leaf shrivelling strain.

(5) Vector Studies.

Tomato aphid populations were studied under field conditions to determine the species colonising tomato plants with a view to testing such species as vectors of *P.V.Y.l.s.*

(a) Aphid Populations.

The method of estimating aphid populations within tomato plantings followed the method introduced by Davies (1934) and later modified by Bald, Norris and Helson (1950). At the commencement of sampling tomato plantings which were chosen for aphid population studies were in the first month of field development.

(i.) *Estimation I.*—A commercial planting in the Ormiston district was selected for the introductory study. Normal cultural practices, which included the fortnightly application of insecticides (parathion and DDT), were continued during the period of assessment. Nine aphid counts were made at intervals of approximately seven days. Slight variations from the weekly schedule were necessary because of weather conditions.

The insecticidal applications restricted or prevented the normal multiplication of aphids. The alate estimations therefore exclude the alate progeny of infesting species. Of the total aphids (1,796) counted during the period of study, 98 per cent. were alate or nymphal forms of *Aphis gossypii;* 15 per cent. of the total forms of this species were alatae and the remainder were recently deposited nymphs; apterae were absent and nymphs did not develop. The remaining 2 per cent. of the total aphid counts comprised alate, apterous and nymphal forms of *Myzus persicae* (0·8 per cent.), *Macrosiphum solanifolii* (0·7 per cent.), and a number of undetermined species. *Aphis gossypii* constituted 94 per cent. of the total alatae.

(ii.) *Estimation II.*—In an experimental tomato planting located at the Domain, Brisbane, tomato leaf samples were collected on four occasions at weekly intervals. Insecticides were not applied to the tomato plants before or during the aphid estimations; this allowed all species, where possible, to colonise the plants. Of the total aphids (2,568) present, 55·6 per cent. were *Myzus persicae,* 30·9 per cent. *Aphis gossypii.,* 10·4 per cent. *Macrosiphum solanifolii* and the remaining 3·0 per cent. undetermined species. The total alatae included 58 per cent. *Aphis gossypii.*

Table 4 summarises the aphid counts for the two tomato plantings. The absence of apterous *Aphis gossypii* in the field counts at Ormiston and the Domain, together with the inability of *A. gossypii* to colonise caged tomato plants growing outdoors in the pots, indicate that the tomato is an unsuitable host for this species. Infestations of *Myzus persicae* and *Macrosiphum solanifolii* in field and potted plants effectively colonised the tomato.

Summarising the aphid data, it was found that during the late autumn and early winter *Aphis gossypii* was the dominant species migrating from external hosts into the two field plantings; *Myzus persicae* and to a lesser extent *Macrosiphum solanifolii* entered plantings in smaller numbers and these species were largely responsible for the colonisation of the one planting at the Domain.

Table 4.

APHID POPULATION COUNTS.

Location.	Leaf Distribution.*	Number of Leaves.	*Aphis gossypii.*				*Myzus persicae.*				*Macrosiphum solanifolii.*				Other Species.				Grand Total.
			Alatae.	Apterae.	Nymphs.	Total.	Alatae.	Apterae.	Nymphs.	Total.	Alatae.	Apterae.	Nymphs.	Total.	Alatae.	Apterae.	Nymphs.	Total.	
Ormiston—																			
Mar. 22, 1955, to May 23, 1955	I.	208	37	..	704	741	1	2	2	5	..	1	..	1	..	1	1	2	749
	II.	208	70	..	458	528	1	..	1	2	..	..	..	..	2	..	..	2	532
	III.	208	81	..	291	372	3	..	..	3	..	..	..	..	3	..	..	3	378
	IV.	208	68	..	50	118	4	1	..	5	1	8	4	13	1	..	..	1	137
	Total	832	256	..	1,503	1,759	9	3	3	15	1	9	4	14	6	1	1	8	1,796
Domain—																			
June 28, 1955, to July 30, 1955	I.	100	89	..	67	156	60	67	317	444	5	8	40	53	3	..	10	13	666
	II.	100	160	..	90	250	84	33	333	450	3	1	30	34	17	1	20	38	772
	III.	100	182	..	82	264	105	19	263	387	2	8	31	41	13	..	7	20	712
	IV.	100	99	..	25	124	74	4	69	147	..	7	133	140	5	..	2	7	418
	Total	400	530	..	264	794	323	123	982	1,428	10	24	234	268	28	1	39	78	2,568

* I., basal leaf; II., ground canopy leaf; III., middle leaf; IV., top leaf (recently expanded).

(b) Aphid Transmission.

Experiments in the glasshouse were conducted to test *Myzus persicae*, *Macrosiphum solanifolii* and *Aphis gossypii* as vectors of *P.V.Y.l.s.*

Method.—Non-infective colonies of *Mysus persicae, Macrosiphum solanifolii* and *Aphis gossypii* were raised on swede turnip, common sow-thistle (*Sonchus oleraceus* L.) and cotton plants respectively. Aphids were subjected to a starvation period followed by an acquisition period and then were transferred by camel-hair brush to one of paired test seedlings of either *Nicotiana glutinosa* or *Physalis floridana* for a transmission period. At the same time, aphids from non-infective colonies were transferred to the second paired seedling for control purposes. At the completion of the transmission period, aphids were removed and the plants sprayed with parathion.

Table 5.

APHID TRANSMISSION.

Mean Daily Temperature* (°F.).	Species.	Number per Test Plant.	Period.			Results.
			Starvation.	Acquisition.	Transmission.	
68	*Myzus persicae*	10 apterae	1 hr.	10 min.	4 hr.	10/10
75	*Myzus persicae*	10 alatae and apterae	..	5 days	24 hr.	5/10
76	*Myzus persicae*	5 alatae	2 hr.	10–15 min.	4 hr.	8/10
78	*Myzus persicae*	3 apterae or nymphs	1 hr.	5 min.	30 min.	4/5
86	*Myzus persicae*	5 alatae	1 hr.	5 min.	30 min.	0/5
		5 alatae	,,	,,	1 hr.	0/5
		5 alatae	,,	,,	2 hr.	0/5
		5 alatae	,,	,,	4 hr.	0/5
		5 alatae	,,	,,	6 hr.	0/5
		5 alatae	,,	,,	24 hr.	0/5
75	*Macrosiphum solanifolii*	10 alatae and apterae	..	5 days	24 hr.	3/10
75	*Aphis gossypii*	10 alatae and apterae	..	5 days	24 hr.	0/8
77	*Aphis gossypii*	5 alatae	1 hr.	5 min.	30 min.	0/5
		3 apterae	1 hr.	5 min.	30 min.	0/5
79	*Aphis gossypii* †	1 alatae	..	..	24 hr.	1/4

* Over 7-day period following aphid transfer to test plants.

† Collected from naturally-infected tomato plant.

The aphid transmission experiments showed that when mean temperatures for the seven days following aphid transfers to test plants were below 80 deg. F., *Myzus persicae* (22/35)* readily transmitted the virus, *Macrosiphum solanifolii* (3/10) transmitted it less readily, and in the case of *Aphis gossypii* (1/19) only occasional individuals transmitted it. When mean temperatures were above 80 deg. F., alate *Myzus persicae* (0/30) did not appear to transmit the virus to test plants. The results of seven experiments are summarised in Table 5.

(6) Field Spread of the Disease.

Observations of the incidence and dispersal of tomato leaf shrivel disease during the 1954-56 surveys of diseased plantings indicated that virus dissemination was due principally to active vectors. Mechanical transference during pruning and other cultural operations appeared to contribute to a lesser degree. The importance of the aphid vectors in dispersing *P.V.Y.l.s.* was recognised during the early investigations by the rapid spread of the disease. The increasing incidence of the disease invariably reached total crop infection prior to the time of harvest, irrespective of the system of tomato management (Officers of the Horticulture Branch 1953).

An introductory study of the mode and rate of virus spread in field plots was conducted during the late autumn-winter period of 1956 at Redlands Experiment Station, Ormiston. Although systematic field data on seasonal variations in tomato aphid populations are not available, the trial provided some useful information on disease incidence and dispersal.

(a) Observation Plots.

Plants of a tomato variety of German origin which has provided an excellent indicator of early virus infection (Fig. 9) were raised in an insect-proofed glasshouse and transplanted into two field plots on Mar. 28, 1956. The plants, 272 in number, were regularly spaced in each plot. One plot was sprayed bi-weekly with parathion and nicotine sulphate alternately to eliminate aphid colonisation and to restrict the movement of alatae within the plot. The second plot, identical in design, remained unsprayed. The plots were separated by a hessian windbreak 6 ft. high which was so placed as not to prevent external alate flights from entering either plot by the agency of wind. Plants exhibiting early symptoms were recorded at bi-weekly intervals. Field charts showing the site of diseased plants, together with the date of infection, were prepared.

(b) Infection.

Total infection in the unsprayed and sprayed plots was attained in 22 days and 37 days respectively. Infection curves were prepared for each area by plotting progressive totals of diseased plants against time. Both the observed

* Numerator denotes infected plants and the denominator denotes paired seedlings.

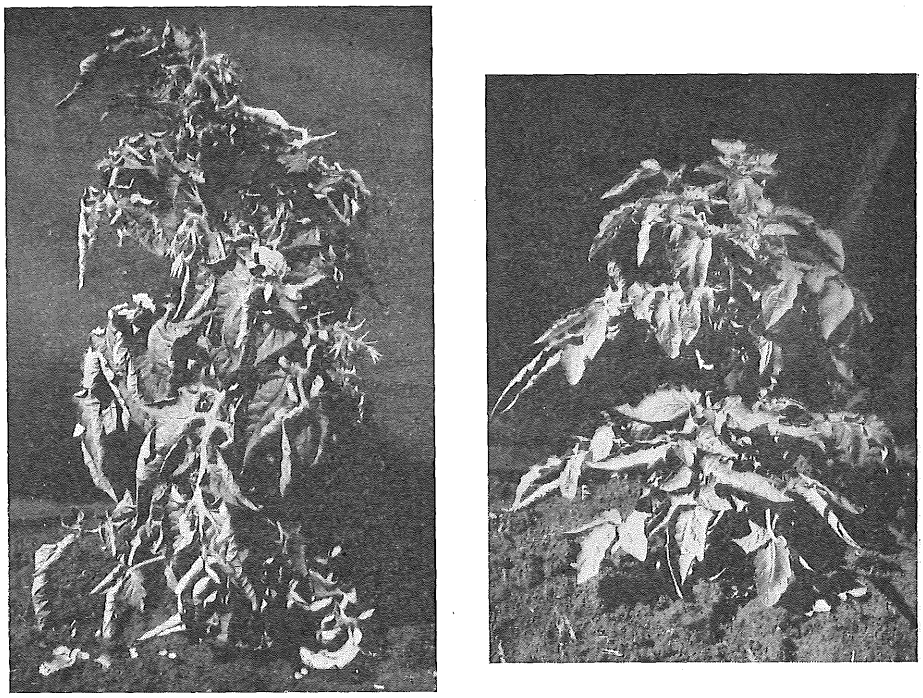

Fig. 9.

Effect of *P.V.L.l.s.* on a Tomato Variety of German Origin. Right, healthy plant. Left, symptom picture in the early stages of infection.

time of early symptom expression and the calculated time of infection based on a time lag of 14 days between infection and expression of symptoms are given in Fig. 10.

(c) Distribution of Diseased Plants.

Initially, the distribution of the diseased plants in both the sprayed and the unsprayed areas on Apr. 19 and Apr. 23 was examined. Though counts were taken on later dates, no statistical analyses were carried out on these because only a small number of plants remained healthy.

Whether the distribution of infected plants could be regarded as random was examined by dividing the sprayed and unsprayed areas into small groups containing 6 plants (2 rows, 3 plants per row) and testing by means of the X^2 test.

In each area there were 44 groups. The expected and observed distributions were as given in Table 6.

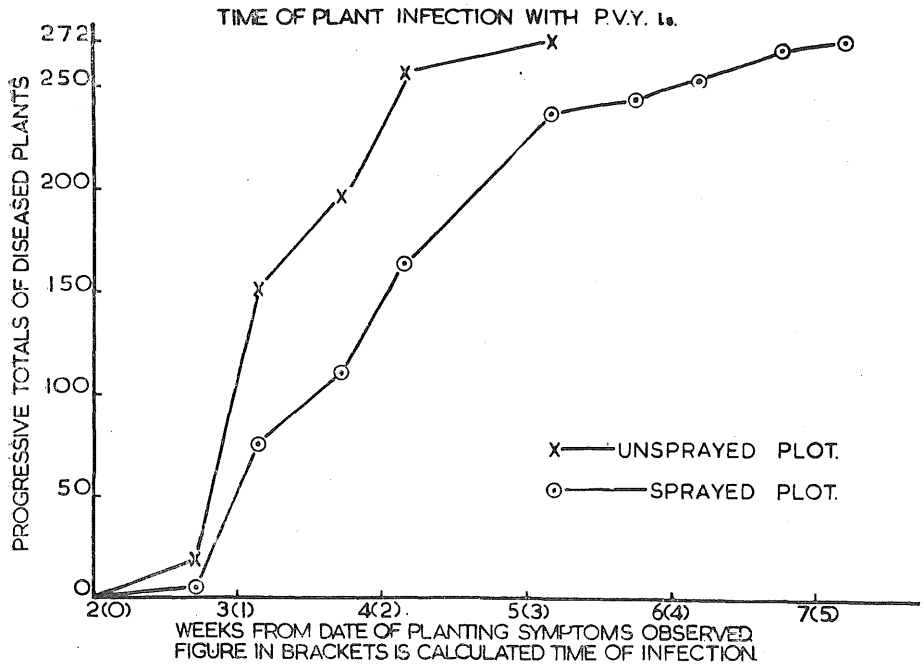

Fig. 10.

Field Spread Trial, 1956. Spread of *P.V.Y.ls.* within sprayed and unsprayed tomato plots.

Table 6.

DISTRIBUTION OF EARLY FIELD INFECTION.

Number Infected.	Sprayed Area.				Unsprayed Area.			
	April 19.		April 23.		April 19.		April 23.	
	Expected.	Observed.	Expected.	Observed.	Expected.	Observed.	Expected.	Observed.
0	6·06	4	9·91	11				
1	14·19	19			10·73	7		
2	13·83	12	13·56	16			11·17	12
3			12·32	9	13·23	19		
4					12·46	13	14·25	13
5	9·82	9	8·21	8			13·35	15
6					7·57	4	5·21	4
X^2	2·64 (not signif.)		1·46 (not signif.)		5·52 (not signif.)		0·65 (not signif.)	
Probability of Infection	0·2803		0·4053		0·5568		0·7008	

For each of the four series the expected and observed distributions showed good agreement, indicating that the distributions in both areas and at the two dates may be regarded as random.

The variation in the number of infected plants per group was analysed into rows, columns and remainder, which was taken as error. Prior to the analyses being made, the $\sqrt{\times + \frac{1}{2}}$ transformation was used in order to equalise the variances.

At each date and for each area, the differences between rows and columns were not significant and no trends across or down the field were observed. Also, no evidence of infection being higher in the 26 border groups than in the 18 interior groups was found.

The main point of interest in studying the relationship between plants diseased between Apr. 19 and Apr. 23 and plants diseased at Apr. 19 was whether the incidence of infection or the proportion of plants infected in the period Apr. 19 to Apr. 23 is higher in the vicinity of a plant infected at Apr. 19 than elsewhere in the field. The plants which were healthy at Apr. 19 were classified into those in the neighbourhood of a plant previously infected and those not so situated. These two groups were then further divided into those which became diseased between Apr. 19 and Apr. 23 and those which were still healthy at the end of the period. The results given in Table 7 were obtained.

To test whether the incidence of disease was higher in the neighbourhood of a diseased plant or not, X^2 was calculated. For the sprayed area $X^2c = 6 \cdot 14$, which is highly significant. In the case of the unsprayed area it is not significant, but the number of healthy plants remaining at this time was so small that any significance is difficult to obtain. It would appear that in both the sprayed and the unsprayed areas the incidence of disease was higher in the neighbourhood of a previously diseased plant.

Table 7.

RELATIONSHIP OF SUBSEQUENT INFECTION TO DISEASED PLANTS.

Plant Position.	Sprayed Area.				Unsprayed Area.			
	Diseased.	Healthy.	Total.	Observed Percentage Diseased.	Diseased.	Healthy.	Total.	Observed Percentage Diseased.
Near a diseased plant	21	60	81	26	36	69	105	34
Not near a diseased plant	14	100	114	12	2	12	14	14
Total	35	160	195	..	38	81	119	..

The random distribution of diseased plants within each plot, 3-4 weeks after transplanting, was consistent with a hypothetical dissemination of the virus by infective aphid vectors migrating from external hosts to tomato plants within the trial. The presence of a diseased plant in any one defined group predisposing neighbouring plants to subsequent infection indicated short flights by infective alate vectors from infected plants to others in the vicinity.

During the period of disease dispersal, estimations of the aphids in each plot were not made owing to the likelihood of mechanical transference of the virus whilst sampling the leaves of each plot. At the time when total infection was reached in the trial, aphid numbers in each plot were checked by the leaf sampling technique as modified by Bald, Norris and Helson (1950). Aphids were absent from the sprayed plot, except for two alate *Aphis gossypii* (100 leaves). Small numbers of aphids, including *Macrosiphum solanifolii, Myzus persicae, Aphis gossypii* and undetermined species, were estimated on the sampled leaves from the unsprayed plot together with evidence of larger populations prior to that date. The influence of insecticide treatment on the random flights of infective aphids as they moved through the sprayed plot was such as to appreciably retard the spread of the virus (Fig. 10).

Summarising the results of the field and glasshouse investigations which pertain to the field spread of the virus by aphid vectors, it is concluded that *P.V.Y.l.s.* enters tomato crops by the three aphid vectors, *Myzus persicae, Macrosiphum solanifolii* and *Aphis gossypii,* establishing foci of infection. During the early stages of crop development, further spread of the virus from foci of infection to other healthy plants in the vicinity or situated at random within the crop is effected by broken short flights of infective vectors. In the following stages of the crop development, spread of the virus is augmented by internal plant colonies of infected *Myzus persicae* and *Macrosiphum solanifolii; Aphis gossypii* finds the tomato an uncongenial host.

Field spread of the virus also takes place within diseased plants by mechanical transference during pruning and other cultural operations. Its rapid dispersal by aphid vectors during the early stages of crop development swamps or conceals the rate of spread by mechanical methods.

(d) Alternative Hosts.

Two alternative weed hosts of the virus commonly found growing in areas surrounding tomato plantings were *Solanum nigrum* and *Nicandra physaloides.* Volunteer plants of the tomato and cape gooseberry (*Physalis peruviana*) were further sources of the virus. At the present stage of the investigations, the significance of alternative hosts of the virus in providing a source of inoculum during the early autumn is not fully understood.

It was demonstrated in the glasshouse that infected stock plants of *Nicotiana glutinosa* and *Physalis floridana* were able to carry the virus without symptoms for up to three months when the mean daily temperature was above

85 deg. F. This would suggest that summer tomato plantings may act as symptomless carriers of the virus. If aphid vectors are able to transmit the virus during periods of high temperature, summer plantings would ensure the carry-over of *P.V.Y.l.s.*

In the absence of facilities to control glasshouse temperatures at or below 70 deg. F. during the summer months, it was not possible to demonstrate by routine glasshouse tests the presence of *P.V.Y.l.s.* in field plantings at this time, or whether aphids are capable of transmitting the virus from one summer planting to another when day temperatures are high. Without such data it is not possible to account, with certainly, for the rapid and extensive appearance of the virus symptoms in the late autumn plantings, but it is considered that the high incidence of the disease in the late autumn cannot be fully explained by the presence of infected alternative hosts as the only source of inoculum.

(7) Economic Significance of the Disease.

For many years the poor setting and development of tomato fruit in south-eastern Queensland, particularly during the winter months, has been a serious problem. Prior to 1955, this problem was attributed to the unsuitability of commercial tomato varieties to local conditions. Evidence obtained from a study of diseased tomato plants which were artificially and naturally infected with *P.V.Y.l.s.* shows that the heavy losses in tomato production were the result of infection with tomato leaf shrivel disease.

(a) Yields of Inoculated Plants.

During the period June–November, 1955, a tomato inoculation-yield trial was conducted within an insect-proofed glasshouse with a soil base.

Method.—Tomato seedlings of the variety Q2 were transplanted into plots in a randomised block design. Each plot contained three plants, which were pruned to one stem and supported by wooden stakes. Protection against plant diseases and insect pests was afforded by fortnightly applications of combined fungicidal and insecticidal sprays. There were six replications of four treatments, consisting of plant inoculations at the first, second and third flowering stages of plant development, and the uninoculated check plants. Virus inoculum was applied to the plants when the majority of plants within any one treatment had reached the appropriate stage of flowering. Progressive totals of ripe fruit numbers and weights from each plant were recorded. At the end of the experiment plants were severed from their root systems at ground level. Each plant was cut into small sections and reduced to a constant dry weight in a hot-air oven.

Progressive totals of the ripe fruit harvested from the four treatments are plotted in Fig. 11. The graph illustrates the serious effects of the disease on the yields of inoculated tomato plants. One important factor influencing the production of diseased plants was found to be the prevailing temperatures. A study of the glasshouse temperature data, recorded by a thermograph throughout the experiment, showed that the mean daily temperature during the early stages of setting and development of fruit was at or near the critical

temperature limit of 75 deg. F. for disease expression. The harvesting of ripe fruit from every plot commenced on Sept. 28 at a time when the mean daily temperature was beyond the critical limit. Ripe fruit which were harvested from Oct. 24 to the end of the trial on Nov. 16 were set prior to or during September at a time when diseased plants were subjected to temperatures which masked the disease.

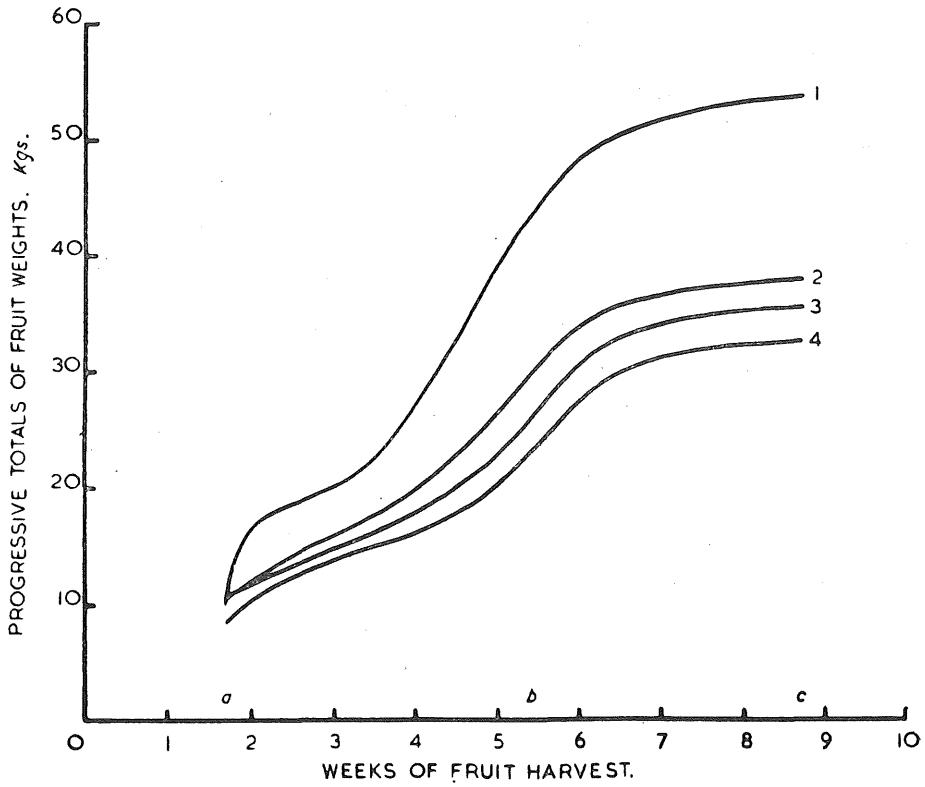

Fig. 11.

Glasshouse Yield Trial, 1955. Progressive totals of ripe fruit from healthy and diseased tomato plants. 1, from healthy plants. 2, 3, 4, from diseased plants inoculated with *P.V.Y.l.s.* at the third, second and first flowering stages, respectively. a-b, early period of fruit harvest. b-c, late period of fruit harvest.

A summary of the treatment means in terms of total fruit weight, numbers and average weight of ripe fruit are shown in Table 8. During the main period of fruit harvest from Sept. 28 to Oct. 24 the yields of diseased plants were significantly reduced. The order of reduction of virus treatments when expressed as a percentage of the yields of healthy plants ranged from 31·1 per cent. to 50·5 per cent. according to the time of inoculation. An analysis of the yield data for the late period of harvest—i.e., from Oct. 24 to

Table 8.

YIELD DATA FOR INOCULATED PLANTS.

Treatment.	Plot Means from Sept. 28 to Oct. 24.			Plot Means from Oct. 24 to Nov. 16.			Plot Means from Sept. 28 to Nov. 16.		
	Yield of Ripe Fruit*.	Number of Fruit*.	Average Weight of Fruit*.	Yield of Ripe Fruit.	Number of Fruit.	Average Weight of Fruit.	Yield of Ripe Fruit*.	Number of Fruit*.	Average Weight of Fruit*.
	g.		g.	g.		g.	g.		g.
Uninoculated	7,770	61·7	127	1,948	21·3	91·7	9,720	83·0	117·2
Inoculation at First Flowering ..	3,850 (50·5)	33·8 (45·2)	114 (10·3)	1,814	19·8	93·0	5,660 (41·8)	53·7 (35·4)	106·5 (9·2)
Inoculation at Second Flowering ..	4,020 (48·3)	35·2 (43·0)	114 (10·3)	2,064	24·2	89·0	6,090 (37·4)	59·3 (28·6)	103·2 (12·0)
Inoculation at Third Flowering ..	4,810 (38·1)	43·7 (29·2)	112 (12·0)	1,364	15·5	87·0	6,180 (36·5)	59·2 (28·7)	105·2 (10·3)
Necessary Differences for Significance—									
5 per cent. level	1,530	12·3	9·3	N.S.	N.S.	N.S.	1,710	15·4	9·8
1 per cent. level	2,110	17·0	12·8	N.S.	N.S.	N.S.	2,360	21·3	13·6

* The figures in brackets represent the percentage reduction compared with control plants.

Nov. 16—showed that the small yield differences between diseased and healthy treatments were not significant. It is construed that the increasing temperatures prior to and during the late period of harvest inhibited the systematic effects of the virus within diseased plants. The differences between treatments for the overall period of harvest, although reduced by the temperature influence during the late harvest, were still significant.

The non-significant differences between the yields of the three virus treatments, irrespective of the period of fruit harvest as shown in Table 8, were attributed to a wide variation in seedling growth at transplanting time. The subsequent variation in flowering did not permit a statistical differentiation of the effects of the disease between virus treatments, as virus inoculum was applied to all plants within any one treatment when the majority of plants within that treatment had reached the specified stage of flowering.

The experimental data on the effects of the disease on plant growth, assessed in terms of plant heights (Table 9) and plant dry weights (Table 10), verified the field observations of the disease. These observations had indicated a substantial reduction in the size of recurved leaves and thickness of stems and no reduction in the distance between leaf nodes.

Table 9.

EFFECT OF DISEASE ON PLANT HEIGHT.

Treatment.	Plot Means.	Reduction.
	In.	Per cent.
Control	225.5	..
First Flowering Inoculation	225·1	0·3
Second Flowering Inoculation	220·3	2·4
Third Flowering Inoculation	218·0	3·4

No significant differences.

Table 10.

EFFECT OF DISEASE ON DRY WEIGHT OF PLANTS.

Treatment.	Plot Means.	Reduction.
	g.	Per cent.
Control	472·8	..
First Flowering Inoculation	274·3	42·0
Second Flowering Inoculation	295·8	37·5
Third Flowering Inoculation	330·7	30·1
Grand Mean	343·4	

Necessary differences for significance 61·8 (5%), 85·4 (1%).

Ripe fruit weights and numbers up to Oct. 28 for each plant were significantly correlated with its final dry weight. The correlation coefficients were 0·78 and 0·80 respectively. Therefore, losses in numbers and weight of ripe fruit were directly related to the reduced growth of diseased plants.

(b) Yields of Naturally Infected Plants.

The yields of 50 tomato plants, which were selected from a field chart showing diseased plant locations and dates of *P.V.Y.l.s.* infection within the field experiment described on page 195, were recorded in terms of numbers and weight of fruit. Ten plants at each of five dates of infection were chosen at random.

An analysis of the yield data (Table 11) showed that there were significant differences in plant yields between the early and the late dates of infection. For each week that plant infection was delayed there was a 6·5 per cent. increase in the number of fruit and a 10·0 per cent. increase in fruit weight.

Table 11.

TIME OF PLANT INFECTION AND YIELD.

Date of Infection.	Number of Fruit.		Yield.	
	Observed Mean.	Estimated Mean.	Observed Mean.	Estimated Mean.
			Lb.	Lb.
April 19	25·9	23·9	2·99	2·87
April 26	24·2	25·8	3·22	3·23
May 3	26·4	27·7	3·37	3·60
May 10	28·7	29·6	3·97	3·96
May 17	33·2	31·5	4·44	4·33
Mean	27·7		3·60	
Necessary differences for significance—				
5 per cent. level	4·8		0·71	
1 per cent. level	6·4		0·95	

Regression coefficient of number of fruit on time of infection = 1·91 fruit per week.

Regression coefficient of yield on time of infection = 0·366 lb. per week.

It was shown previously for this planting in Fig. 9 that bi-weekly applications of insecticides to a field plot of 272 tomato plants appreciably retarded the spread of tomato leaf shrivel disease as compared with an unsprayed plot. In this experiment bulk weighings of the two plots were recorded (Table 12). The fruit yields of the sprayed plot was increased by 60·2 per cent.

Table 12.

EFFECT OF DELAYED DISEASE SPREAD ON YIELD.

Plot (272 plants).	Number of Fruit.	Yield.
		Lb.
Unsprayed	4,763	509
Sprayed	7,338	876
Percentage increase by spraying	54·0	60·2

This study of naturally infected plants confirmed the importance of the time of plant infection in conditioning the final yields of diseased plants which was not significantly expressed in the glasshouse trial.

(c) Discussion.

Under field conditions, it was not possible to demonstrate the yield differences between diseased and healthy tomato plants. This was due to the rapid and uncontrolled spread of *P.V.Y.l.s.* from diseased plants to healthy check plants by the active aphid vectors. The tomato-inoculation experiment in the glasshouse did show that the yields of diseased plants were substantially reduced during the main period of fruit harvest despite the higher temperatures prevailing in the glasshouse.

In south-eastern Queensland, field temperatures from the late autumn to the early spring may fall 15–17 deg. F. below the critical temperature limit (75 deg. F.) for disease expression, as shown in Table 1. Consequently, it may be inferred from the results of the glasshouse trial that a much greater decline in the yields of diseased plants may be expected under the low field temperatures which prevail during this period, as the effects of the disease increase in severity with decreasing temperatures.

(8) Varietal Susceptibility to the Disease.

During the late winter and early summer months of 1955, a number of commercial and introduced tomato varieties, including unnamed selections, were subjected to field infection in two experimental plantings at Ormiston. The varieties under observation were Athens, Caleplate, Cluster Mato, Cuyano, Dunstan, Durbot, Early Garden State, Grosse Lisse, Kopiah, Long Red, Manalee, Manalucie, Market Favourite, Market Supreme, Marman, Marmande, Pearson, Peron, Plamar, Potentate, Q1, Q2, Q3, Q4, Queen, Red Cloud, Rouge de Marmande, Salads Special, Sioux, Sioux (dwarf), Summer Prolific, Tatura Dwarf Globe, Thesoloniki, Valiant, Vokal, Western Red and Wisconsin 55. Varietal reactions based on plant growth and size and number of fruit suggested that Grosse Lisse, Queen, Q2, Manalucie, Red Cloud, Valiant and Wisconsin 55, although far from virus-tolerant, were the least susceptible varieties.

The prevailing temperatures in the glasshouse, even during the coolest months of the year modified the reaction of susceptible diseased varieties to such an extent that a macroscopic assessment of individual plants or varieties was impossible. Glasshouse experiments were therefore conducted under mean daily temperatures ranging from 70 deg. to 72 deg. F. to evaluate other methods of assessing tomato varieties with respect to their susceptibility, tolerance or immunity to the disease. One experimental technique, which was based on the numerical assessment of the diseased and healthy plants in terms of their relative dry weights, is described below.

Method.—Seedlings were raised and transplanted at the 4-leaf stage into clay pots containing 4 lb. of air-dried, steam-sterilized soil. One seedling was transplanted into each pot. Two weeks later, plants within each variety were paired in quintuplicate according to equal plant growth. In descending order of growth, one pair of each variety was randomised in a block layout. One plant of each pair was mechanically inoculated with *P.V.Y.l.s.* The inoculated and healthy plants within each pair were also randomised. At the completion of the experiment, plants were reduced to a constant dry weight in a hot-air oven.

Twenty-three tomato varieties and selections, species and interspecific crosses within the genus *Lycopersicon* were tested by the above technique in two experiments, with results as given in Tables 13 and 14 respectively. In each experiment a trend in virus susceptibility or tolerance was indicated, although no significant differences were obtained. The variety Q2, common to each experiment, was constant in its reaction to infection. Within a number of the

Table 13.

MEAN VALUES FOR GROWTH AND MEAN GROWTH REDUCTION OF INOCULATED PLANTS, EXPERIMENT I.

Variety.	Total Growth.		Growth Reduction.	
	Inoculated Plants.	Healthy Plants.	Mean.	Equivalent Mean*.
	g.	g.	Per cent.	Per cent.
Pearson	12·76	17·91	31·7	27·7
Valiant	10·59	15·05	31·8	27·8
Potentate	13·22	18·92	32·6	29·0
Prosperity	13·12	19·89	35·1	33·0
Salads Special	9·69	15·01	35·2	33·3
Bowen Buckeye	10·05	15·79	36·8	35·8
Q2	9·29	14·77	36·8	35·9
Break o' Day	8·13	12·88	36·9	36·0
Marglobe	9·37	15·04	37·7	37·3
South Australian Red Dwarf	7·83	12·74	38·0	37·9
Sioux	9·00	15·09	38·9	39·4
Rouge de Marmande.. ..	6·59	13·05	44·7	49·4
	Healthy plants > > inoculated plants		No significant differences	

* Inverse sine transformations.

Table 14.

MEAN VALUES FOR GROWTH AND MEAN GROWTH REDUCTION OF INOCULATED PLANTS, EXPERIMENT II.

Variety, Selection and Species.	Total Growth.		Growth Reduction.	
	Inoculated Plants.	Healthy Plants.	Mean.	Equivalent Mean.
	g.	g.	Per cent.	Per cent.
Manalucie	1·69	2·16	8·8	2·4
Ohio Wilt Resistant Globe ..	2·09	2·63	10·9	3·6
((Grosse Lisse x (Grosse Lisse x *L. pimpinellifolium*)) x *L. hirsutum* (*tetraploid*)	1·18	1·51	14·7	6·4
L. pimpinellifolium	0·93	1·11	14·7	6·5
Simi	1·87	2·41	16·9	8·5
Rey de los Tempranos—(diploid)	2·50	3·19	17·6	9·1
147–3 (bulk line)	1·25	1·82	19·3	11·0
Rey de los Tempranos—(autotetraploid)	1·62	2·15	23·1	15·4
Stambovoi	0·80	1·09	25·3	18·2
147–3 (single line)	2·60	3·57	27·1	20·7
L. peruvianum	1·46	2·28	36·1	34·6
Q2	2·08	3·32	36·9	36·0
Healthy >> inoculated plants (overall).				
For varieties 147–3 (single line) and Q2, healthy >> inoculated plants.			No significant differences.	
For species *L. peruvianum*, healthy > inoculated plants.				

selections, varieties and interspecific crosses, a wide range in variability was recorded. This was attributed to genetic variation within segregating lines and technical difficulties in pairing small seedlings according to growth. Without further refinement the technique used is not considered a reliable method for gauging varietal reaction to the virus infection.

In Queensland, the testing of plants under controlled temperatures below 65 deg. F. offers the simplest method of determining varieties or individual plants with the necessary levels of tolerance or immunity to *P.V.Y.l.s.* Inoculated plants, susceptible to the virus, are readily determined by virtue of their necrotic reaction and severe growth reduction when growing under conditions of low temperature.

II. TOMATO YELLOW SHRIVEL DISEASE.

A virus disease of the tomato which is commonly referred to as the tomato yellow shrivel disease has been widespread in the coastal districts of eastern Queensland for a number of years. Recent studies showed that the disease resulted from a dual virus infection. One component of the virus complex was the leaf shrivelling strain of *potato virus Y* and the second component was the aucuba strain of *tobacco mosaic virus* (*T.M.V.a.*).

(1) History and Field Distribution.

The history and field distribution of tomato yellow shrivel disease is closely linked with that of the tomato leaf shrivel disease. The latter disease has been recognised since 1948, while records of the Department of Agriculture and Stock show that the aucuba or yellow mosaic disease of the tomato has been widespread in this State since 1932. Tomato yellow shrivel disease has been recorded in the Bowen, Mackay, Rockhampton, Nambour and Brisbane districts of Queensland. Tomato specimens from these districts gave positive reactions for *P.V.Y.l.s.* and *T.M.V.a.* when sap extracts from diseased plants were inoculated into differential hosts growing in the glasshouse. A similar disease has been described by Conover and Fulton (1953) in Florida and by MacNeil (1955) in Ontario.

(2) Symptoms.

The virus complex was isolated from a number of tomato plants which exhibited a wide and varied pattern of symptoms. Symptoms which are known to be associated with *T.M.V.a.* were found to dominate the disease expression, although foliar symptoms are sufficiently distinctive to render infected plants prominent in a field. Such factors as prevailing temperatures, the order of plant infection by the two viruses and the stage of plant development contributed to the observed variations in symptom expression.

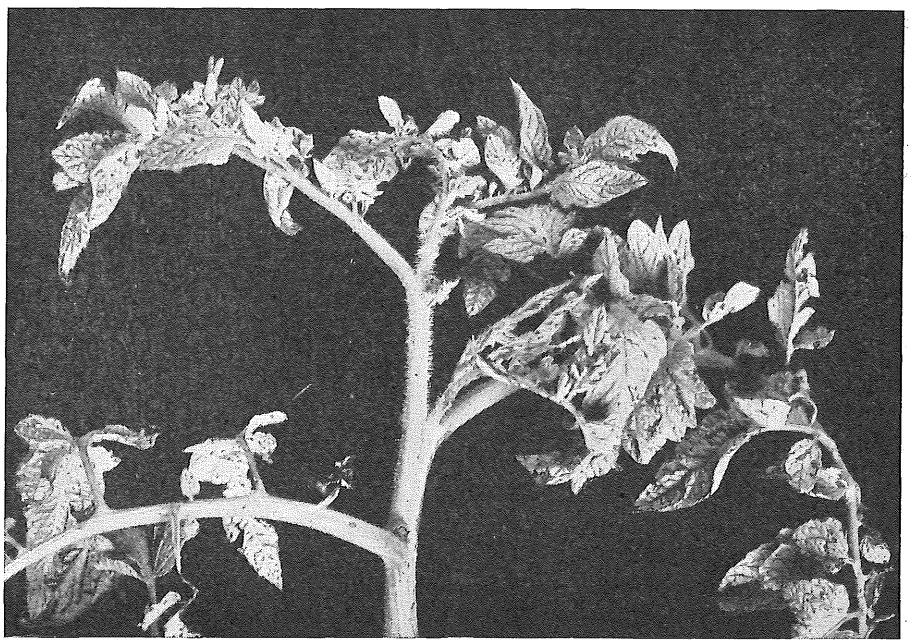

Fig. 12.

Terminal Symptoms of Tomato Yellow Leaf Shrivel Disease, Showing Variable Necrotic Spotting and Lightening of the Foliage.

From spring to early summer the typical expression of the disease (Figs. 12–14) may be summarised as follows:—

Systematic yellowing and recurving downwards and inwards of leaves and leaflets; variable spot and marginal leaf necrosis (Fig. 12); variable green vein banding or interveinal yellowing of the expanded leaves; variable vein purpling on the under-surfaces of leaves; necrotic streaks on stems and petioles; extensive shrivelling of the lowest leaves which rapidly advances upwards; shrivelled leaves remaining attached to the stem; internal vascular and pith necrosis of stem tissues commencing in the lower region of the stem; variable necrosis associated with the developing fruit; flowering poor or absent; cessation of fruit-setting; growth stunting.

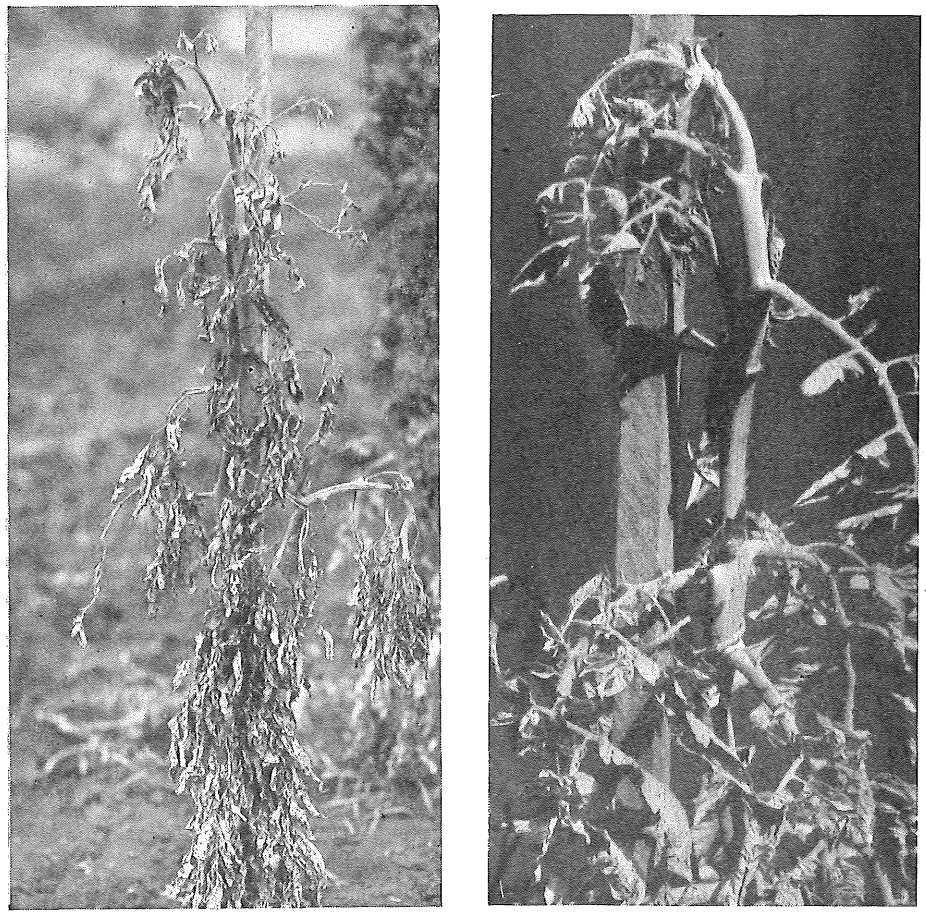

Fig. 13.

Terminal Wilting (right) and Final Death (left) of Tomato Plant Due to *P.V.Y.l.s.–T.M.V.a.* complex.

Fig. 14.

Symptoms of *T.M.V.* and *P.V.Y.l.s.—T.M.V.a.* Complex on Tomato. Right, var. Q2 inoculated with *T.M.V.* Left, companion plant inoculated with *P.V.Y.l.s.—T.M.V.a.* complex, showing stunting and commencement of leaf shrivel.

The variations in the terminal symptoms of the disease range from a mosaic pattern of light-green, yellow and dark-green to a bright yellow rugosity with variable spot necrosis, or to an upward rolling of small, dark-green, rim-bound leaflets with the presence of an intense vein purpling on the leaflet under-surfaces. The fully expanded leaves of diseased plants may exhibit varying degrees of yellowing, necrosis, mosaic pattern, blistering and leaf shrivelling.

In certain cases, diseased plants become flaccid and express symptoms of a wilt condition (Fig. 13). The root and stem tissues of such plants, when sectioned, show an internal vascular browning or necrosis. The tissues also

develop, in extreme cases, a localised or systemic necrosis of the pith which extends through to the stem surface (Fig. 15). The necrosis appears on the surface as a girdling sunken lesion. Diseased plants with the extensive internal stem necrosis gradually enter a state of permanent wilt and finally collapse (Fig. 13).

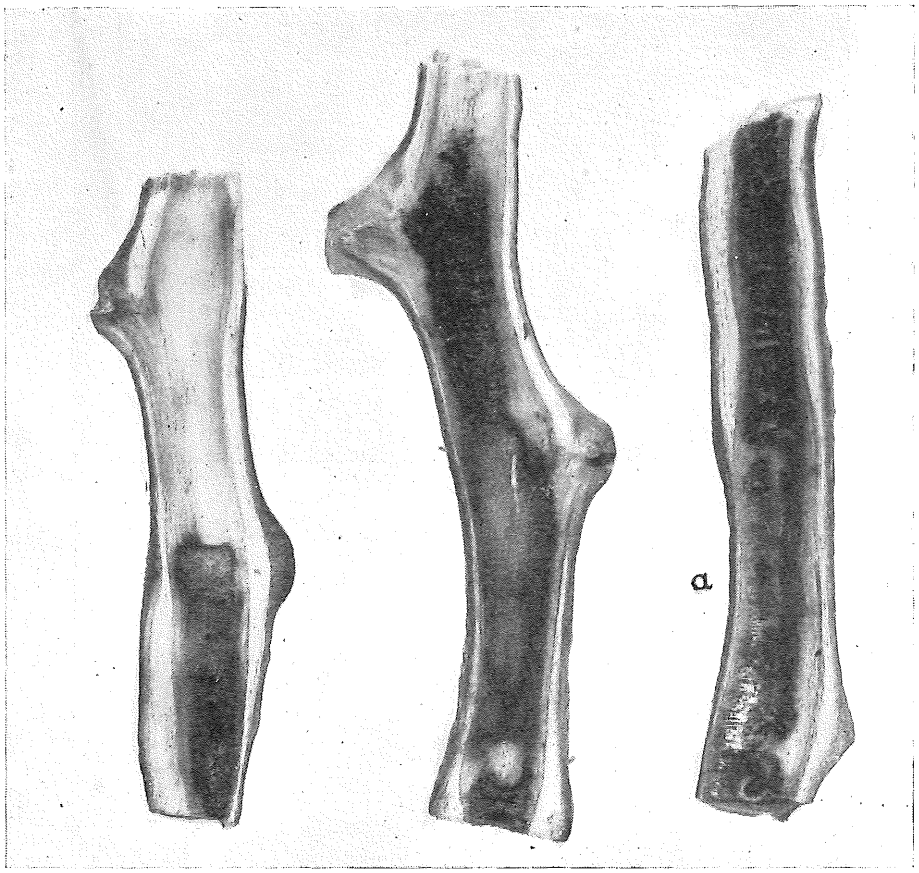

Fig. 15.

Longitudinal Sections of Stems of Tomato Infected with *P.V.Y.l.s.-T.M.V.a.* Complex, Showing Internal Browning and Necrosis which has Extended to the Surface at a.

Occasionally the terminal growth of diseased plants exhibits a shoe-string symptom which has been described for aucuba (yellow) tomato mosaic disease. Usually only two or three leaves express this symptom.

The effect of the virus complex on fruit production is more serious than the effect of tomato leaf shrivel disease. The fruit set of plants infected with the virus complex at or prior to the first flowering stage of plant development

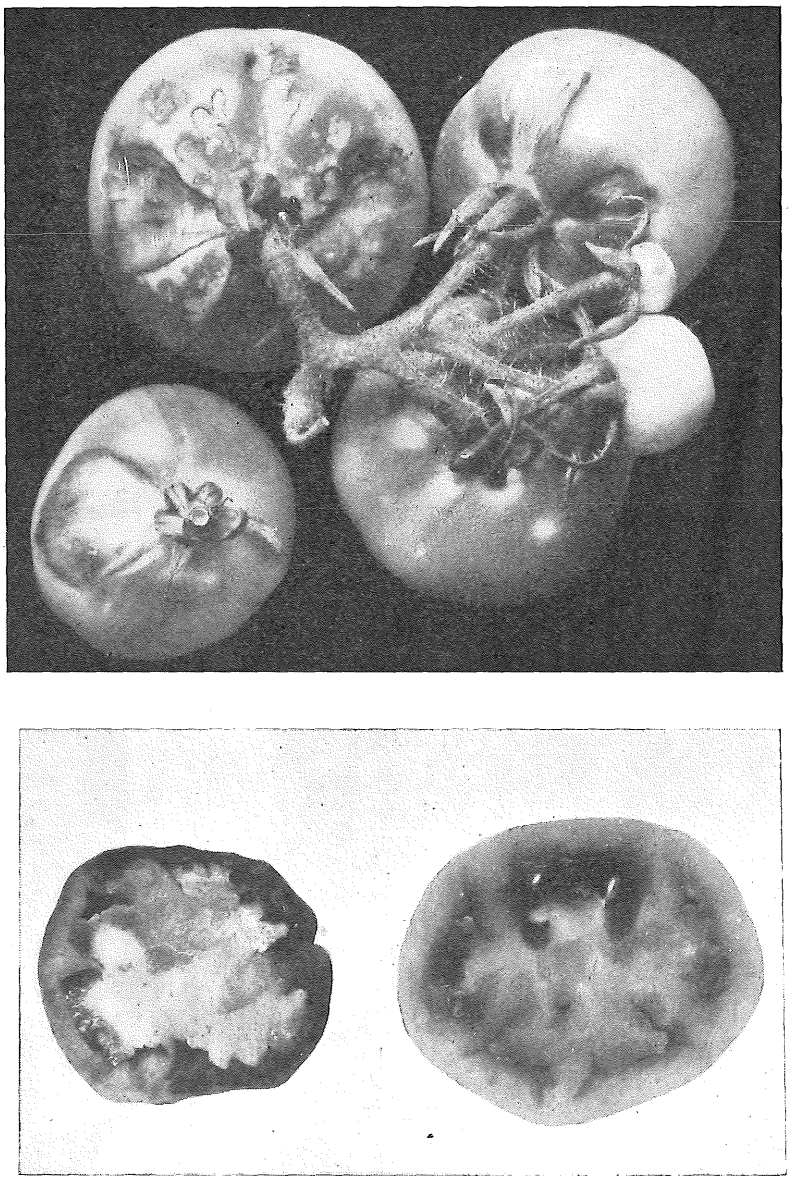

Fig. 16.

Tomato Fruit from Plants Naturally Infected with *P.V.Y.l.s.–T.M.V.a.* Complex, Showing External and Internal Necrosis.

is generally nil. Many fruit which are set prior to the dual virus infection develop necrotic lesions on the surface, usually near the stalk end. The necrosis extends into the pulp cavity (Fig. 16). Also the fruit, on ripening, are often marked by yellowish blotches.

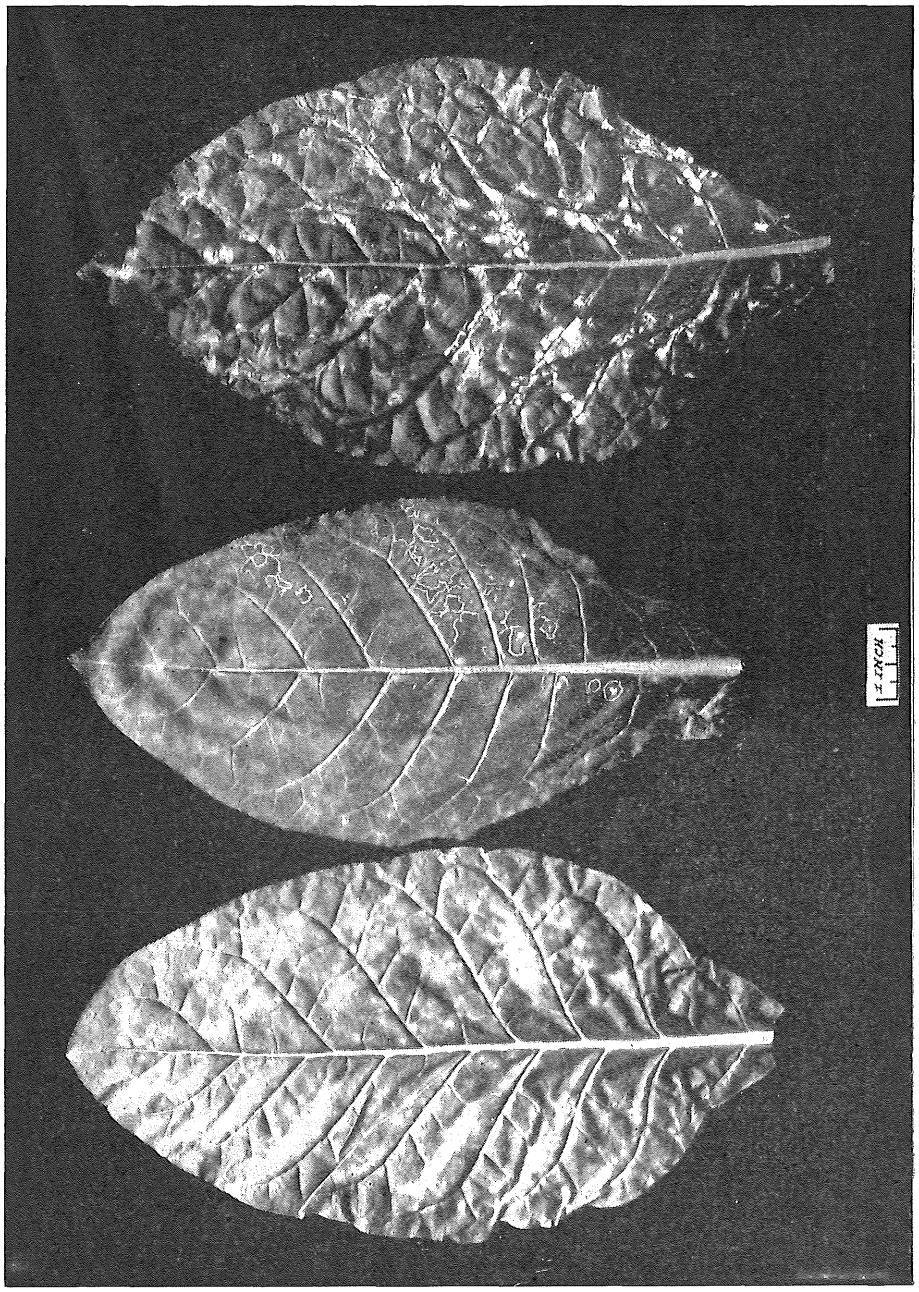

Fig. 17.

Local Reactions of Tobacco Leaves Inoculated With *P.V.Y.l.s.–T.M.V.a.* Complex. Bottom, White Burley showing chlorotic spotting. Centre, Smyrna showing necrotic line and ring spot pattern. Top, Hickory Pryor showing necrotic lesions and traces of vein necrosis.

(3) Host Reactions.

The main feature of the virus complex distinguishing it from *P.V.Y.l.s.* was the production of a severe necrotic phase. This again was conditioned by prevailing temperatures. The following solanaceous species which were mechanically inoculated with the virus complex exhibited varied localised and necrotic systemic symptoms under mean temperatures ranging from 79 deg. to 80·5 deg.

Nicotiana tabacum.—

Two tobacco varieties (Hickory Pryor and Smyrna) expressed necrotic symptoms, whilst a third variety (White Burley) did not (Fig. 17).

Hickory Prior.—Numerous local necrotic spots which coalesced to form large necrotic lesions; vein necrosis; systemic stem necrosis commencing at the point of attachment of inoculated leaves; terminal vein clearing; mild leaf mottle; mild green vein banding; secondary necrotic spotting on the lower leaves.

Smyrna.—Local chlorotic spots; necrotic line pattern surrounding chlorotic spots; terminal yellow-green and dark-green mosaic pattern or leaf mottle; slight interveinal puckering; secondary line necrosis (ring-spot pattern) on lower leaves.

White Burley.—Local chlorotic spots; coalescence to form large chlorotic areas; terminal vein clearing; chlorotic areas; yellow-green and dark-green mosaic pattern; raised blisters; leaf distortion; reduction in leaf lamina.

Nicotiana glutinosa.—

Numerous local necrotic lesions; coalescence of lesions; leaf shrivelling; stem necrosis at the point of attachment of inoculated leaves; necrotic sunken lesion girdling the stem; terminal vein clearing; chlorosis; leaf distortion; slow necrotic collapse and variable plant recovery from axillary side-shoots below stem lesion.

Nicotiana sylvestris.—

Numerous local grey-brown necrotic lesions which coalesce to form large necrotic lesions; shrivelling of inoculated leaves; necrotic stem lesion formed where inoculated leaves are attached to stem; terminal vein clearing, interveinal puckering, leaf reduction and distortion, variable variegated necrotic line pattern; chlorotic mottle or green distorting mosaic pattern with dark-green raised blisters; secondary necrosis, yellowing and shrivelling of lower leaves.

Datura stramonium.—

Numerous local small necrotic lesions; variable vein necrosis; small necrotic stem lesions at the point of attachment of inoculated leaves; abscission of inoculated leaves; small seedlings may develop a systemic necrosis.

Petunia hybrida.—

Local necrotic and chlorotic lesions; variable terminal vein clearing; interveinal chlorosis; ballooning of interveinal tissues; mild leaf distortion; systemic stem and plant necrosis.

Nicandra physaloides.—

Isolated local necrotic and chlorotic spots; vein necrosis; abscission of inoculated leaves; terminal leaf distortion; light-green and dark-green mottle; interveinal puckering; secondary vein necrosis; necrotic streaks on stems; chronic leaf abscission; breaking of flower colour; severe internal stem necrosis; slow systemic plant necrosis.

Solanum melongena—

Few local necrotic red-brown lesions surrounded by chlorotic halo; systemic vein necrosis; shrivelling of inoculated leaves; variable stem necrosis at the point of attachment of inoculated leaves.

(4) Resolution of the Virus Complex.

Four techniques were employed to separate *T.M.V.a* and *P.V.Y.l.s.* from the virus complex.

(1) *P.V.Y.l.s.* has a thermal inactivation point ranging from 56 deg. to 60 deg. C. (page 188) and *T.M.V.a.* has a thermal inactivation point of 90 deg. C. One portion of the expressed sap from naturally infected tomato plants was heated to 70 deg. C. for 10 min. The heated sap was inoculated into 10 plants of *Nicotiana glutinosa.* The untreated portion of the sap was inoculated into a second series of 10 plants of *N. glutinosa.* All inoculated plants in both series developed a local necrotic reaction, which indicated the presence of *T.M.V.a.* The plants inoculated with the untreated sap developed in addition a systemic terminal reaction. Further inoculations into indicator plants by expressed sap from plants in each series confirmed that *P.V.Y.l.s.* had been inactivated by the heat treatment. Severin (1950) used a similar technique to separate *T.M.V.* (*t*) from western cucumber mosaic virus.

(2) Johnson and Valleau (1935) showed that *T.M.V.* survives for more than 50 years in desiccated material. *P.V.Y.l.s.* has been shown to remain infectious within desiccated leaves for a period not exceeding eight days. Accordingly, leaves were detached from tobacco plants which had been inoculated with the virus complex and retained between blotting paper for periods up to 22 months. The desiccated tobacco leaves were macerated in a buffer solution. Extracts were mechanically inoculated into 10 plants of *Nicotiana glutinosa* and *Nicotiana sylvestris.* Further tests confirmed the presence of *T.M.V.a.* and the inactivation of *P.V.Y.l.s.*

(3) Non-infective apterous forms of *Myzus persicae* from stock colonies were starved for one hour in petri dishes. The aphids were then given an infection feed for 30 min. on diseased tomato leaves infected with the virus complex. Twenty aphids were transferred to each of 10 plants of *Nicotiana glutinosa.* All plants were infected by *P.V.Y.l.s.* and not by *T.M.V.a.* A second experiment, which was conducted along similar lines, confirmed this separation.

(4) When mean daily temperatures in the glasshouse were below 70 deg. F., plants of *Nicotiana glutinosa* which were inoculated with the virus complex acted as filter plants. *T.M.V.a.* was localised in the leaves and *P.V.Y.l.s.* spread systemically through the plants. The latter was recovered from the terminal growth of infected plants by sub-transfers to a further series of plants. *P.V.Y.l.s.* was separated in this way from the complex on three separate occasions. The virus separation was not affected when mean daily temperatures were above 80 deg. F. Under these temperature conditions the virus complex became systemic.

(5) Identity of the Components.

P.V.Y.l.s. was identified by the routine methods described earlier. The identity of *T.M.V.a.* was confirmed by inoculations into a range of indicator plants (*Nicotiana tabacum, Nicotiana glutinosa, Nicotiana sylvestris, Datura stramonium, Solanum melongena, Physalis floridana, Petunia hybrida, Vigna sinensis, Cucumis sativus, Cucurbita pepo, Citrullus vulgaris* and *Zinnia elegans* L.); the thermal inactivation point; and the stability of the virus in desiccated tissue. The local necrotic reaction of the isolates on *Nicotiana sylvestris* determined the strain identity (Kunkel 1934).

(6) Field Spread of the Disease.

Field observations of tomato plantings in south-eastern Queensland showed that plant infections by *P.V.Y.l.s.* generally preceded infections by *T.M.V.a. P.V.Y.l.s.* mainly enters tomato crops by active aphid vectors, which are further responsible for the internal dissemination of the virus from foci of infection. The second component of the virus complex, *T.M.V.a.*, gains entry either from contaminated soil, in the seedbed or the field (Berkeley 1942), from contaminated hands, clothes and implements, and from external plant reservoirs of the virus (Smith 1937). Internal spread from foci of infection takes place at the various times of handling. Tomato yellow shrivel disease spreads rapidly in staked, trellised and cradled crops during the early phase of crop development, while in ground crops the disease tends to be spread largely during harvest operations. Market garden areas which grow tomato crops annually during the autumn, winter and spring months are the most seriously affected areas.

(7) Economic Significance of the Disease.

An important aspect of tomato yellow shrivel disease is its serious effects on fruit production. In a field experiment 32 staked tomato plants of the variety Q2 growing under field conditions were mechanically inoculated with the virus complex at the first flowering stage of plant development. Ten plants failed to set any fruit and the remaining 22 plants averaged less than two fruit per plant. Evidence indicated that such fruit had been set at the time

of inoculation and subsequent fruit set was absent. In commercial plantings, where the virus complex was observed to enter during the early stages of plant development, total fruit loss resulted.

Tomato yellow shrivel disease is regarded as a potential threat to tomato production in market garden areas, where regular handling of plants is practised. From the late autumn to the early summer, most tomato plantings are totally infected with *P.V.Y.l.s.* by the time of fruit harvest. The appearance and severity of tomato yellow shrivel disease in these crops is dependent on the stage of crop development at the time of entry of *T.M.V.*

III. TOMATO FERN-LEAF SHRIVEL DISEASE.

During surveys of tomato plantings, a number of diseased tomato plants which exhibited both fern-leaf and leaf shrivelling symptoms were encountered. Transfers of expressed sap from diseased plants to a number of solanaceous and cucurbit hosts in the glasshouse established the presence of a virus complex. Field isolates of the disease were resolved into two viruses. One component was *cucumber mosaic virus* (*C.M.V.*), which causes the tomato fern-leaf disease, and the other was the leaf shrivelling strain of *potato virus Y.*

The disease has been detected in tomato plantings within the Domain, Raby Bay, Cleveland, Ormiston, Birkdale and Aspley districts surrounding Brisbane. The seasonal occurrence of the disease was restricted to the low-temperature months of late winter and early spring, which is in keeping with the studies by Mogendorff (1930) on the effects of temperature on the expression of tomato fern-leaf disease.

(1) Symptoms.

Preliminary observations of tomato fern-leaf shrivel disease have indicated that, apart from the shrivelling of the lower leaves, the disease conforms to the symptoms of tomato fern-leaf (Mogendorff 1930). As with tomato leaf shrivel disease, shrivelling of the lower leaves advances systematically upwards.

(2) Resolution of the Virus Complex.

C.M.V. was readily separated from the virus complex by transferring field isolates of the disease, which were retained on stock plants (*Nicotiana tabacum* and *Nicotiana glutinosa*) to two cucurbit hosts—*Cucumis sativus* var. Early Fortune and *Cucurbita pepo.* var. Long Cream Running. It had been shown previously (page 185) that these cucurbit hosts are immune to *P.V.Y.l.s.*

It was found that over the summer months in the glasshouse, the *C.M.V.* component was inactivated by retaining stock plants inoculated with the virus complex under the high temperature conditions (Table 1). During the following winter months, *P.V.Y.l.s.* was recovered from such plants but not *C.M.V.*

(3) Identity of the Virus Components.

The resolved virus components were identified by their symptom expression when sap extracts of each component were mechanically inoculated into a range of indicator plants.

(a) C.M.V. Component.

Indicator plants exhibited the following reactions when they were mechanically inoculated with *C.M.V.*:—

Cucumis sativus var. Early Fortune:—Local chlorotic lesions. Systemic rugose yellow and dark-green mosaic patterns.

Cucurbita pepo var. Long Cream Running:—Local chlorotic lesions. Systemic yellow spot mottle.

Citrullus vulgaris var. Hawkesbury Wilt Resistant:—No reaction.

Pisum sativum var. Dwarf Greenfeast:—Local necrotic lesions and death of inoculated leaves. No systemic reaction.

Vigna sinensis var. Black Eye:—Local and small red lesions surrounded by chlorotic halo. No systemic reaction.

Phaseolus vulgaris var. Black Beauty:—No reaction.

Zinnia elegans var. Lilliput:—Systemic vein clearing; chlorosis; scattered dark-green areas; mild leaf distortion.

Lycopersicon esculentum var. Q2:—Systemic vein clearing; filiform leaflets; twisting and distortion of leaves.

Datura stramonium:—Systemic vein clearing; chlorotic areas; irregular white line pattern.

Nicotiana glutinosa:—Local chlorotic areas. Systemic vein clearing; chlorosis; white line patterns; interveinal puckering.

Nicotiana tabacum:—Systemic vein clearing; mild light-green and dark-green mottle; interveinal puckering.

With certain variations, the symptoms of *C.M.V.* expressed by the above hosts conformed to the symptoms described for other strains of *C.M.V.* (Ainsworth 1935, Pound and Walker 1948, Fulton 1950, Bhargava 1951, Doolittle and Zaumeyer 1953).

(b) P.V.Y.l.s. Component.

The second component separated from the virus complex conformed to the reactions of the field isolates of tomato leaf shrivel disease when sap extracts were mechanically inoculated into a range of differential hosts.

(4) Field Spread and Economic Importance.

Aphid vectors of *P.V.Y.l.s.* are also vectors of *C.M.V.* (Smith 1937, Bawden 1950). During August and September the virus complex spreads rapidly to reach total crop infection. This rapid spread is attributed to the aphid vectors common to the two viruses. The tomato fern-leaf shrivel disease was found to be restricted to a few plantings in the districts surrounding Brisbane. In young plantings the disease was responsible for total fruit loss.

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