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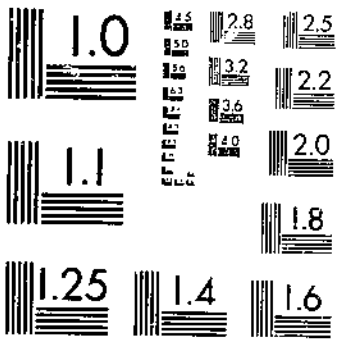

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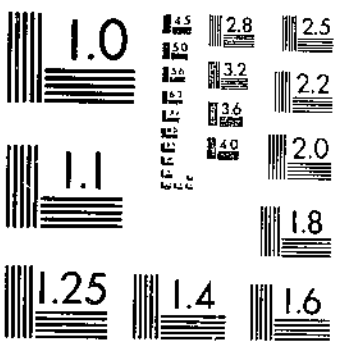

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Sugar Beet

YELLOWS DISEASE

in the United States

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Sugar Beet Yellows Disease in the United States

By C. W. Bennett, *plant pathologist, Crops Research Division, Agricultural Research Service*[1]

INTRODUCTION

The disease of sugar beet, now called virus yellows, probably was first known in Europe under the name "Jaunisse." It was described by Quanjer (*41*)[2] in 1934, who suspected that it was caused by a virus. This was confirmed by Van Schreven (*51*) and Roland (*44*) in 1936, who showed that the causal virus was transmitted by aphids. In England in 1935 Petherbridge and Stirrup (*40*) described the disease and designated it as "virus yellows," to distinguish it from other types of yellowing. They stated that the causal virus was transmitted most commonly through the agency of *Aphis fabae* Scop. and that the disease had been present for some years both in England and on the Continent. Watson (*54*) identified the disease in England in 1938, at which time it was common throughout all of the sugar-beet-producing districts.

Yellows was first positively identified in the United States in 1951 by Coons and Kotila (*17*), at which time it already had a wide distribution. The studies, results of which are reported herein, were begun at the U.S. Field Station, Riverside, Calif., in 1951 and continued there and at the U.S. Agricultural Research Station in Salinas, Calif., from 1951 to 1959, inclusive.

The virus used in these studies is considered to be unrelated to radish yellows virus, described by Duffus (*21*), which causes yellowing difficult to distinguish from yellowing induced by the less virulent strains of beet yellows virus.

ORIGIN AND GEOGRAPHIC DISTRIBUTION OF YELLOWS

Little is known of the origin and geographic spread of the yellows disease of sugar beet. This is owing in part to the fact that yellowing

[1] The writer wishes to acknowledge his indebtedness to Phyllis R. Emparan of the U.S. Agricultural Research Station, Salinas, Calif., for assistance with all phases of the greenhouse tests from 1954 to 1958, inclusive; and to R. C. Dickson, University of California, at Riverside, and to Norman F. McCalley, formerly of the University of California, at Davis, for verification of the identification of aphids used in the insect transmission tests.

[2] Italic numbers in parentheses refer to Literature Cited, p. 60.

of older leaves, which is the most obvious symptom, often occurs late in the season and has been attributed to natural ripening of the plants or to soil or other environmental conditions.

The disease has no doubt existed in Europe, and perhaps elsewhere, for many years. Gram (*26*) presented evidence that virus yellows has existed in Denmark at least since 1914, although it was not identified as a distinct virus disease until much later.

Schlösser (*48*) postulated that the yellows virus originated in wild beets, possibly *Beta maritima* L., in England, spread to cultivated forms, and was carried to the Continent by aphids borne on favorable winds. There is little evidence to substantiate this theory, however, and it seems probable that the origin of the disease cannot now be traced, owing to the fact that it was widely distributed before its effects were recognized as a distinct disease.

The date of first appearance of the disease in the United States and the source of virus for initial infection also have not been determined.

Although yellows in the United States is similar to or identical with that in Europe, it is not known that the causal virus was introduced into this country from Europe. The disease was not identified with certainty in the United States until 1951, but Kodachrome slides, supplied by Roy Bardin of the Monterey County Department of Agriculture, California, indicate strongly that it was present in the Salinas Valley as early as 1945. The disease may have been present much earlier. Records of the Salinas factory of a commercial sugar company from 1921 to 1959 show no obvious break in either sucrose percentage or tonnage that would indicate introduction of yellows virus during that period. However, tonnage has increased irregularly, but sucrose percent has gradually decreased.

On the other hand, there is evidence that the disease has spread to new areas in the past few years. The sugar beet industry in the Imperial Valley of California, which was started in 1938, was apparently free of yellows up to and including 1956. In 1957 diseased plants were observed in several fields in March, and by the middle of April infection had become widespread. The disease was prevalent again in 1958 and in 1959. This area, however, has a considerable degree of isolation, and delay in the introduction of the virus might be expected.

Regardless of its origin, the yellows disease is now found in nearly all countries where the sugar beet is grown. It has been reported from the British Isles, Spain, France, the Netherlands, Denmark, Norway, Sweden, Germany, Italy, Austria, Hungary, Yugoslavia, U.S.S.R., Turkey, Iran, Syria, Australia, and the United States.

In the United States the disease has been found in all States where the sugar beet is grown commercially. However, the disease has been severe only in those States in which beets, or other susceptible plants, live throughout the year. The disease occurs most extensively in California, Oregon, Washington, and Colorado. It has been reported from Kansas and Nebraska, but its importance in these States is not clearly defined. It was discovered in seed fields in the Salt River Valley of Arizona in 1955. Diseased plants have been found also in Idaho, Utah, Ohio, Minnesota, and Michigan, but apparently yellows has not caused measurable losses in these States.

HOST RANGE OF THE YELLOWS VIRUS

For several years after the yellows disease was described, the causal virus was considered to have a rather restricted host range. More recently, the range of known susceptible species has been greatly expanded (Bennett and Costa (*6*); Canova (*13*); Schlösser, Fuchs, and Beiss (*50*); Kristensen (*34*); Roland (*45*); and Bercks and Zimmer (*9*)). A list of species, reported as susceptible, was compiled by Zimmer (*61*) in 1956. Further host-range studies were reported by Björling (*11*) in 1958.

Observations and tests in the United States have indicated that, as a rule, only a limited number of species of plants are infected extensively under field conditions. Transmission tests, however, have shown that the virus has a very wide potential host range and that many of the common weeds in Western United States are susceptible to infection under greenhouse conditions.

Plants Found Infected in the Field

Yellows attacks sugar beets, table beet, Swiss chard, spinach, and New Zealand spinach in areas in Western United States where the disease occurs. Yellows has been found also on some of the common weed plants in the field. These include *Chenopodium album* (lambsquarters), *C. murale* (nettleleaf goosefoot), *Amaranthus retroflexus* (redroot amaranth), *Senecio vulgaris* (common groundsel), and *Atriplex semibaccata* (Australian saltbush). A high incidence of infection has been observed on plants of *C. album* in the early spring in spinach fields. Usually, however, infection is low even in locations where there is an abundance of inoculum. *C. murale* is a common weed in beetfields in the Salinas Valley and other areas of California; however, plants very rarely show symptoms of yellows. This is true also of *Amaranthus retroflexus* and *Senecio vulgaris*. *Tetragonia expansa* occurs along the coastal area of central California. Only three plants have been found infected with the yellows virus. No clear symptoms of yellows have been observed on plants of *Atriplex semibaccata* in the field, but tests of plants from the Salinas Valley of California and the Salt River Valley of Arizona have indicated that in both areas many plants are infected. Since *A. semibaccata* is perennial, it seems probable that a high percentage of plants may carry the virus in areas where yellows is prevalent on sugar beet or other susceptible crop plants.

Plants Infected Experimentally

Tests have been made at different times over a period of more than 6 years to obtain additional information on the possible host range of the yellows virus. Potted plants of the species and varieties selected for testing were inoculated by means of large numbers of green peach aphids from sugar beet plants infected with vein-clearing strains of the yellows virus. Inoculated plants were observed for the appearance of symptoms of disease. A month or more after inoculation, nonviruliferous green peach aphids were

allowed to feed on the inoculated plants. These aphids were later transferred to sugar beet or some other susceptible species, to determine whether the yellows virus was present in the inoculated plants. These tests were made with all the species tested, regardless of whether symptoms were evident.

The species of plants shown in these tests to be susceptible to infection by the yellows virus are listed below in alphabetical order with respect to family, genus, and species:

AIZOACEAE

Tetragonia expansa Murr. (New Zealand spinach)

AMARANTHACEAE

Amaranthus albus L. (Rough pigweed)
A. carneus Greene
A. caudatus L. (Love-lies-bleeding)
A. cruentus L. (Purple amaranth)
A. graecizans L. (Tumbleweed amaranth)
A. palmeri S. Wats.
A. retroflexus L. (Redroot amaranth)
Celosia sp.
C. argentea L.
C. cristata L. (Cockscomb)
Gomphrena globosa L. (Globe amaranth)

BORAGINACEAE

Pectocarya pusilla Gray

CARYOPHYLLACEAE

Cerastium viscosum L. (Sticky mouse-ear chickweed)
Gypsophila elegans Bieb. (Babysbreath)
Lychnis coronaria (L.) Desr. (Mullein-pink)
Silene gallica L. (French silene)
S. verecunda S. Wats.
Spergula arvensis L. (Corn spurry)
Stellaria media (L.) Cyr. (Common chickweed)

CHENOPODIACEAE

Atriplex bractcosa S. Wats. (Bract scale)
A. canescens James. (Fourwing saltbush)
A. coronata S. Wats. (Crown saltbush)
A. coulteri Dietr.
A. elegans (Moq.) Dietr. (Wheelscale saltbush)
A. expansa (D. & H.) S. Wats. (Fogweed)
A. hastata L. (Fat-hen)
A. hortensis L. (Garden orach)
A. microcarpa (Benth.) Dietr. (Dot scale)
A. rosea L. (Redscale)
A. semibaccata R. Br. (Australian saltbush)
Bassia hyssopifolia (Pall.) Kuntze. (Fivehook bassia)
Beta atriplicifolia Rouy
B. lomatogona Fisch. & Mey.
B. macrocarpa Guss.
B. maritima L.
B. patellaris Moq.
B. patula Ait.
B. procumbens Chr. Sm.
B. trigyna Waldst. & Kit.
B. vulgaris L. (Sugar beet)
B. vulgaris L. var. *cicla* L. (Swiss chard)
B. webbiana Moq.
Chenopodium album L. (Lambsquarters)
C. amaranticolor Coste & Reyn.
C. ambrosioides L. (Mexican-tea) var. *anthelminticum* L. (Wormseed)
C. capitatum (L.) Asch. (Strawberry-blite)

C. leptophyllum S. Wats. (Slimleaf goosefoot)
C. murale L. (Nettleleaf goosefoot)
C. urbicum L. (City goosefoot)
C. watsoni A. Nels.
Cycloloma atriplicifolium (Spreng.) Coult. (Winged pigweed)
Kochia scoparia (L.) Schrad. (Summer-cypress)
Monolepis nuttaliana (R. & S.) S. Wats.
Salsola kali var. *tenuifolia* Tausch (Russian-thistle)
Spinacia oleracea L. (Spinach)
Suaeda moquini Greene (Alkali sea-blite)

COMPOSITAE

Senecio vulgaris L. (Common groundsel)

CONVOLVULACEAE

Convolvulus occidentalis Gray

PLANTAGINACEAE

Plantago erecta Morris
P. insularis Eastw.

LEGUMINOCEAE

Melilotus indica (L.) All. (Yellow sweetclover)

RESEDACEAE

Reseda odorata L. (Mignonette)

SOLANACEAE

Nicotiana bigelovii S. Wats. (Indian tobacco)
N. clevelandii Gray.

In addition to the species listed above, several species of plants gave indication of susceptibility to infection, but the type of virus with which the plants were inoculated was not recovered. Plants of *Capsella bursa-pastoris* (L.) Medic. produced increased amounts of red pigment on old leaves after inoculation, but they were not noticeably dwarfed. Reactions were essentially the same with all virus strains and transfer back to sugar beet produced very faint symptoms of yellows, or none. Plants of *Samolus parviflorus* Raf. (water-pimpernel) inoculated with yellows virus developed thickened rolled leaves with necrotic spots, and the inoculated plants were dwarfed. Transfer of aphids from such plants to sugar beet gave inconclusive results. Whether the yellows virus is attenuated by passage through these plants remains to be determined. It is suspected that in some cases symptoms noted may have been produced by viruses other than yellows virus or by distinct strains of the yellows virus to which these species are susceptible.

Many species that were inoculated showed no symptoms of yellows and no virus was recovered from inoculated plants. It cannot be stated that all these species are immune from yellows, but it seems probable that, if not immune, they are highly resistant to the strains of the yellows virus used in these tests. Species that showed no symptoms, and from which no virus was recovered, are listed below alphabetically with respect to family, genus, and species.

AIZOACEAE

Mesembryanthemum aequilaterale Haw. (Sea-fig)

APOCYNACEAE

Vinca rosea L. (Periwinkle)

BALSAMINACEAE

Impatiens sp.

BOMBACACEAE

Chorisia speciosa St. Hil. (Floss-silktree)

BORAGINACEAE

Heliotropum curassavicum L. (Salt heliotrope or seaside-heliotrope)
Myosotis arvensis (L.) Hill. (Field forget-me-not)

CARYOPHYLLACEAE

Dianthus barbatus L. (Sweet William)
D. caryophyllus L. (Carnation)
D. chinensis L. (Pinks)
Silene nutans L. (Nodding catchfly)

CHENOPODIACEAE

Atriplex lentiformis (Torr.) S. Wats. (Quailbush)
A. polycarpa (Torr.) S. Wats (Cattle saltbush)
Chenopodium bonus-henricus L. (Good King Henry)

COMPOSITAE

Achillea millefolium L. (Common yarrow)
Ambrosia psilostachya DC. (Western ragweed)
A. trifida L. (Big ragweed.)
Calendula officinalis L. (Pot marigold)
Carthamus tinctorius L. (Safflower)
Cirsium vulgare (Savi) Tenore (Bull thistle)
Eclipta alba (L.) Hassk.
Erigeron sp. (Fleabane)
Gnaphalium bencolens Davidson
Helianthus annuus L. (Common sunflower)
Helichrysum bracteatum Andr. var. *monstrosum* Bailey (Strawflower)
Hypochaeris radicata L. (Hairy catsear)
Lactuca sp.
L. sativa L. (Lettuce)
L. serriola L. (Prickly lettuce)
Parthenium argentatum Gray (Guayule)
Sonchus arvensis L. (Perennial sowthistle)
S. asper (L.) Hill. (Field sowthistle)
S. oleraceus L.
Taraxacum megalorhizon (Forsh.) Hand.-Mazz. (Krim-saghyz)
T. officinale Weber (Dandelion)
Zinnia elegans Jacq. (Zinnia)

CONVOLVULACEAE

Convolvulus arvensis L. (Bindweed)
Ipomoea purpurea (L.) Roth. (Morning-glory)

CRUCIFERACEAE

Brassica nigra (L.) Koch. (Black mustard)
B. oleracea L. var. *gemmifera* D.C. (Brussels sprouts)
B. rapa L. (Turnip)
Cheiranthus cheiri L. (Wallflower)
Dithyrea wislizenii Engelm.
Lepidium lasiocarpum Nutt.
Matthiola incana (L.) R. Br. (Stock)
Raphanus sativus L. (Radish)
Sisymbrium altissimum L. (Tumble-mustard)
S. irio L.

CUCURBITACEAE

Citrullus vulgaris Schrad. (Watermelon)
Cucumis sativus L. (Cucumber)

EUPHORBIACEAE

Eremocarpus setigerus Benth. (Turkey mullein)
Euphorbia maculata L. (Spotted spurge)
E. serpyllifolia Pers. (Thymeleaf spurge)

GERANIACEAE

Geranium dissectum L. (Common geranium)
G. maculatum L. (Spotted geranium)

GESNERIACEAE

Saintpaulia sp. (African-violet)

GRAMINEAE

Avena sativa L. (Oat)
Hordeum vulgare L. (Barley)
Sorghum halepense (L.) Pers. (Johnsongrass)
S. vulgare Pers. (Sorghum)
Zea mays L. var. Country Gentleman. (Corn)

HYDROPHYLLACEAE

Phacelia campanularia Gray. (California bluebell)

LABIATAE

Marrubium vulgare L. (Horehound)
Salvia splendens Ker. (Scarlet sage)

LEGUMINOSAE

Arachis hypogaea L. (Peanut)
Cyamopsis tetragonoloba (L.) Taub. (Guar)
Lathyrus odoratus L. var. Early Flowering Spencer (Sweetpea)
Medicago lupulina L. (Black medic)
M. sativa L. (Alfalfa)
Melilotus alba Desr. (White sweetclover)
M. dentata (Waldst. & Kit.) Pers.
Phaseolus lunatus L. var. *macrocarpus* (Benth.) Van Eselt. (Broadbean)
P. vulgaris L. var. Stringless Greenpod. (Bean)
Pisum sativum L. (Garden pea)
Pueraria lobata (Willd.) Ohwi
Trifolium pratense L. (Red clover)
Vicia sativa L. (Common vetch)
Vigna sinensis (Torner) Savi (Cowpea)

LILIACEAE

Allium cepa L. (Onion)
Tigridia sp. (Tigerflower)
Tulipa sylvestris L. (Tulip)

LINACEAE

Linum lewisii Pursh. (Lewis flax)
L. usitatissimum L. (Flax)

LOASACEAE

Mentzelia pumila (Nutt.) Torr. & Gray (Blazing-star)

LYTHRACEAE

Ammannia coccinea Rottb. (Red-stem)

MALVACEAE

Gossypium hirsutum L. (Cotton)
Hibiscus esculentus L. (Okra)
H. rosa-sinensis L. (Chinese hibiscus)
Malva parviflora L. (Little mallow)
M. rotundifolia L. (Running mallow)
M. sylvestris L. (High mallow)

NYCTAGINACEAE

Mirabilis jalapa L. (Ornamental four-o'clock)

ONAGRACEAE

Epilobium angustifolium L. (Fireweed)
E. paniculatum Nutt. (Autumn willowweed)

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OXALIDACEAE

Oxalis sp.
O. corniculata L. (Creeping woodsorrel)

PAPAVERACEAE

Eschscholtzia californica Cham. (California poppy)
Papaver sp.
P. somniferum L. (Opium poppy)

PHYTOLACCACEAE

Phytolacca americana L. (Pokeweed)

PLANTAGIANCEAE

Plantago coronopus L.
P. lanceolata L. (Buckhorn plantain)
P. major L. (Rippleseed plantain)

POLYGONACEAE

Eriogonum fasciculatum Benth. (Flat-top)
Polygonum acre H.B.K. (Dotted smartweed)
P. argyrocoleon Steud.
Rumex acetosella L. (Sheep sorrel)
R. obtusifolius L. (Bitter dock)
R. persicarioides L. (Golden dock)
R. pulcher L. (Fiddleleaf dock)

PORTULACACEAE

Portulaca oleracea L. (Common purslane)
P. retusa L.

PRIMULACEAE

Anagallis arvensis L. (Scarlet pimpernel)

ROSACEAE

Prunus persica (L.) Batsch. (Peach)
Sanguisorba minor Scop. (Small burnet)

RUTACEAE

Citrus sinesis (L.) Osbeck. (Sweet orange)

SAURURACEAE

Anemopsis californica (Nutt.) Hook. (Yerba mansa)

SCROPHULARIACEAE

Mimulus sp.
Nemesia sp.
Veronica peregrina L. (Neckweed)

SOLANACEAE

Capsicum frutescens L. (Pepper)
Datura meteloides DC. (Tolguacha)
D. stramonium L. (Jimsonweed)

SOLANACEAE

D. tatula L. (Purple thorn-apple)
Hyoscyamus niger L. (Black henbane)
Lycopersicon esculentum Mill. var. New Stone (Tomato)
Nicandra physalodes (L.) Pers. (Apple-of-Peru)
Nicotiana glauca Graham (Tree tobacco)
N. glutinosa L.
N. paniculata L.
N. plumbaginifolia Viv.
N. rustica L. var. Iowa (Wild tobacco)
N. rustica L. var. *pumila* (Wild tobacco)
N. stocktonii Brandeg.
N. sylvestris Speg. & Comes

N. tabacum L. vars. Turkish and Maryland Broadleaf (Tobacco)
Petunia hybrida Vilm. (Petunia)
Physalis wrightii Gray.
Solanum dulcamara L. (Bitter climbing nightshade)
S. elaeagnifolium Cav. (Silverleaf nightshade)
S. nigrum L. (Black nightshade)
S. tuberosum L. (Potato)

TROPAEOLACEAE

Tropaeolum peregrinum L. (Nasturtium)

UMBELLIFERAE

Apium graveolens L. var. *dulce* DC. (Celery)
Conium maculatum L. (Poison hemlock)
Daucus carota L. (Carrot)
Petroselinum crispum (Mill.) Nym. vars. Plain Italian and Smooth Leaf (Parsley)

URTICACEAE

Urtica californica Greene (Coast nettle)

VITACEAE

Vitis sp. (Grape)

ZYGOPHYLLACEAE

Tribulus terrestris L. (Puncturevine)

SYMPTOMS OF THE DISEASE

Symptoms on Sugar Beet

Yellows produces a rather wide range of symptoms on sugar beet, but effects on standard commercial varieties do not appear to vary greatly. A much wider range of symptoms was produced on certain selections from breeding stocks, some of which were very severely damaged by the disease. Environmental conditions influence symptoms. Effects under greenhouse conditions, for instance, may differ in some respects from those found in the field.

Symptoms on Greenhouse Plants

Partly as a result of lower light intensity, yellowing is less marked in the greenhouse than in the field. First symptoms produced by the more virulent strains of yellows virus usually consist of vein clearing, or vein yellowing, in young leaves, beginning as early as 6 days after infection (fig. 1, *A*). The vein effect may be limited to clearing or yellowing of the larger veins, but in some instances it is accompanied by a certain amount of necrosis of cells along the veins, which sometimes produces a type of "etch." In some instances the veins may show considerable necrosis, and in extreme cases almost the entire network of veins of individual leaves may become necrotic.

Growth in veins that show clearing or necrosis is limited, and, as the leaf matures, the interveinal tissue thickens and the veins sometimes appear depressed, particularly on the underside of the leaf. This gives a type of roughness on the lower side of the leaf that has been called "alligator skin," because of its resemblance to the line depressions and irregular-shaped elevations in tanned hides of alligators. The effect, shown in figure 1, *B*, appears to be limited to leaves that have shown vein clearing.

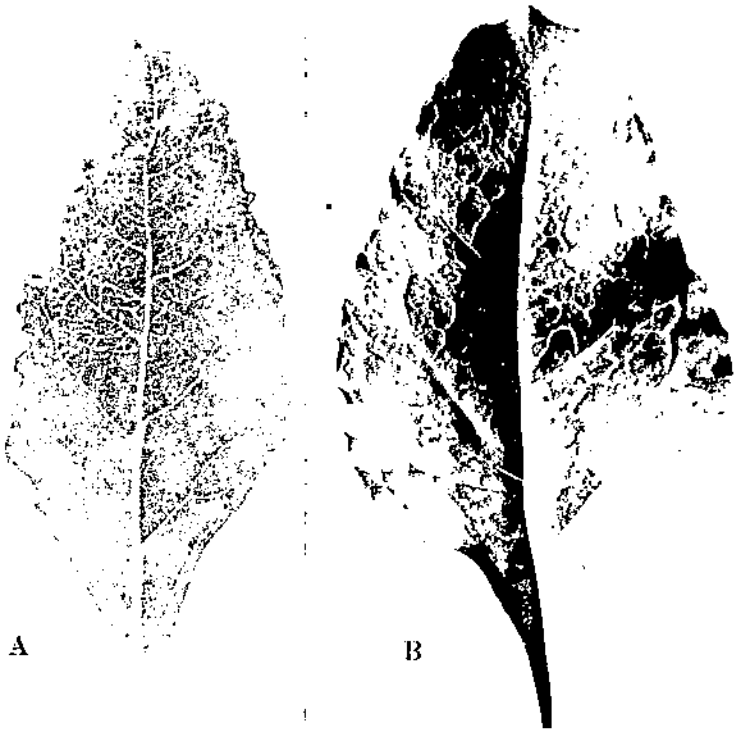

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Figure 1.—**Beet leaves, showing the effects of a virulent strain of yellows virus on young leaves under greenhouse conditions: *A*, Upper side of immature leaf showing vein clearing; *B*, lower side of leaf that earlier had shown vein clearing, but now shows depressed veins resulting from retarded vein development and thickening of interveinal tissue.**

Shortly after vein clearing or yellowing appears, leaves just older than those showing vein clearing, begin to yellow. Entire leaves may yellow more or less uniformly, or certain segments may remain green.

Vein clearing may be found on two to four leaves, but leaves produced later usually are normal until they approach maturity. They may then begin to show yellowing, which may be more or less uniform; but, more often, yellowing may appear in the form of splotches, as shown in figure 2, *A*. In some instances, leaves approaching maturity may show small, translucent, "pinpoint" spots of the type shown in figure 2, *B*. These are usually marked by small elevations on the lower side of the leaf. They appear to be caused by the development of local meristematic areas adjacent to the vascular sheath on the phloem side of the vascular bundle (*35*). The pinpoint symptom has been observed only on leaves of plants in the later or chronic stages of disease and only on plants in the greenhouse.

As leaves of diseased plants age, the tissue between the larger veins usually fades, sometimes leaving the veins green. Older leaves are thickened and brittle, and often die prematurely.

The less virulent strains of virus induce no vein clearing or other obvious effects on young leaves. Symptoms are confined largely

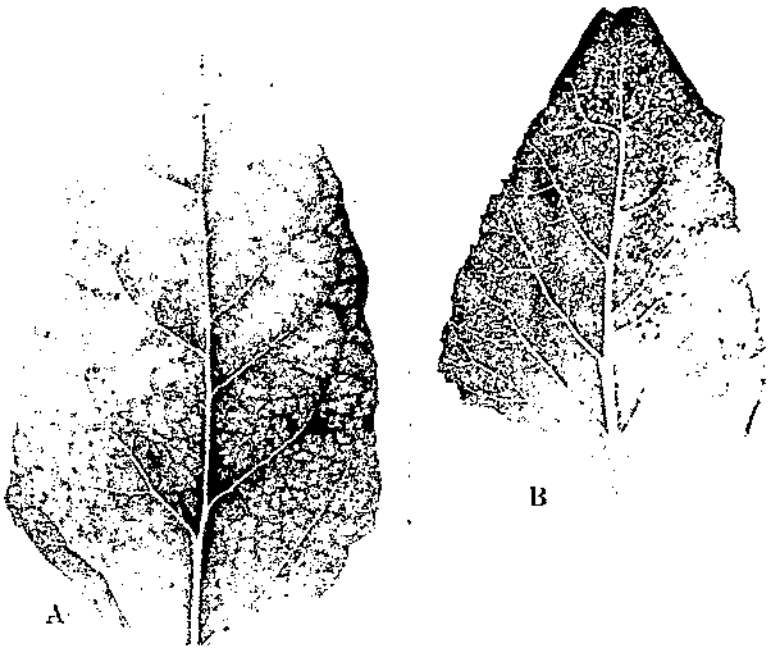

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Figure 2.—Beet leaves, showing effects produced by virulent strains of yellows virus on chronically diseased plants as the leaves approach maturity: *A*, Flecking or mottling, typical of effects frequently observed on leaves of greenhouse plants infected with virulent strains of virus; *B*, "pinpoint" effect, relatively rare under greenhouse conditions and not observed under field conditions.

to older leaves and are similar to those produced on old leaves by the more virulent strains of yellow virus, except that they are less severe.

Symptoms on Plants in the Field

Under conditions of natural spread of yellows in the field in California, first symptoms often appear in late April or early May, 15 to 25 days after plants are infected. Sometimes the disease begins to appear as spots of yellow plants scattered at random throughout the field. These spots represent spread from initial points of infection caused by aphids that came into the field earlier in the season. The disease spreads radially from infected plants and the yellow spots enlarge, often until the entire field is yellow. In some instances, particularly in central California, spread is so extensive and rapid that centers of infection are not evident, or are evident for only a few days, and fields appear to yellow uniformly within a period of 2 to 3 weeks.

The effect of yellows on individual plants varies, depending on the virulence of the strain of virus involved, fertility of the soil, and possibly other factors. As in the greenhouse, first clearly obvious symptoms produced by virulent strains of the virus appear on the young leaves and consist of clearing or yellowing of the veins. Even before vein clearing appears, leaves of the plants may tend to droop slightly.

This condition is apparent only in contrast with the appearance of adjacent healthy plants. Vein clearing may appear on three to five leaves, which in some selections may be dwarfed at maturity. Mature leaves that earlier showed vein clearing often show sunken veins on the lower side of the leaf, which produces a roughened or "alligator skin" effect that is even more evident than on plants in the greenhouse.

About the time of appearance of vein clearing on young leaves, three to five leaves just older may begin to yellow over the entire surface or, more often, in areas sharply delimited by main veins; this type of symptom is sometimes called "sectoring" (fig. 3, *A*). The yellow

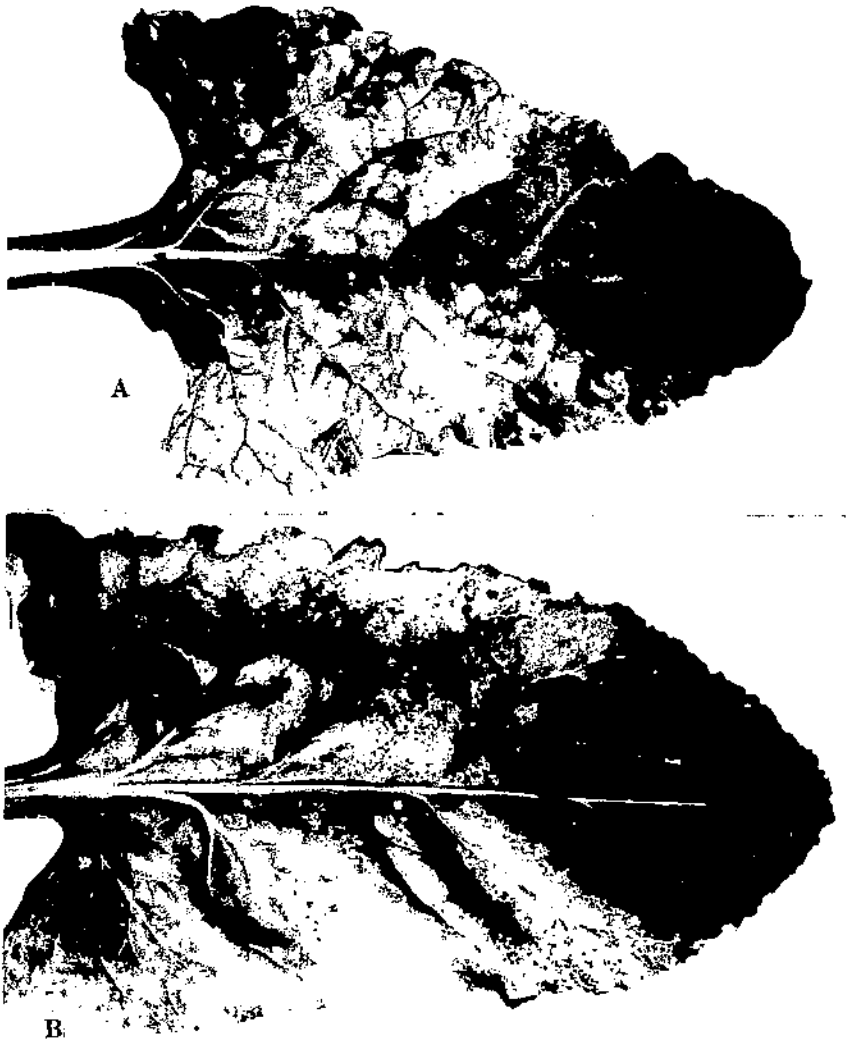

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Figure 3.—A, Beet leaf from field plant, with unequal distribution of symptoms, or "sectoring," often found on the first leaves that show evidence of disease. *B*, Beet leaf similar to that in (*A*), but showing necrosis.

leaves or sectors may show a greater amount of necrosis than leaves produced in the greenhouse and more necrosis than leaves in the chronic stage of disease on the same plant, as illustrated in figure 3, *B*.

Leaves produced after vein clearing appears are more or less normal until they approach maturity. They then may show various types of yellowing that may be more or less uniform, or the yellowing may consist of various types and sizes of ill-defined splotches. Usually, leaves tend to yellow first at the tips and in parts having the greatest exposure to sunlight. As the leaves become older, areas between the larger veins fade, as shown in figure 4, *A*, and sometimes become necrotic. Often necrotic areas are invaded by fungi, resulting in dark spots between the veins, a condition sometimes called "leopard spot" by growers (fig. 4, *B*). Often the larger veins remain green even after the tissue between veins becomes necrotic. Leaves in the later stages of disease are thickened, leathery, and brittle, and die prematurely.

As in the greenhouse, the less virulent strains of the yellows virus do not produce vein clearing on young leaves and first symptoms consist of yellowing of leaves approaching maturity. Otherwise, symptoms are similar to those produced by the more virulent strains.

Although the common commercial varieties of sugar beet do not vary greatly in their reaction to yellows, certain selections from breeding stock have shown a wide range of effects. Some show very little yellowing, whereas others are extremely yellow. A few selections show marked necrosis of mature leaves, which gives the plant a burned appearance. In plants with red pigment, the red color is intensified. Leaves may show various shades of red and in some varieties the leaves have a distinct bronze cast, often in the form of spots that may later become necrotic.

The intensity of both yellowing and necrosis is influenced by the rate of growth of the plants as well as by other factors. In fields fully infected with yellows, plants with a space advantage, such as those in border rows or areas of poor stands, are often greener than plants in other parts of the field. Plants in very fertile soil usually show less yellowing than those in soil of medium fertility. The addition of nitrogenous fertilizers may greatly reduce yellowing.

Symptoms on Crop Plants Other Than Sugar Beet

Beta vulgaris L. (table beet).—Most table beet varieties show essentially the same reactions to the yellows virus as sugar beet, except that leaves of diseased plants are likely to be deep red with little yellowing in evidence. Usually, infected plants in the field may be readily identified by the deep-red color of the foliage. Plants are somewhat dwarfed, and the leaves are thickened and brittle and often become necrotic with age. Leaves on seedstalks are likely to show less reddening and more yellowing than other leaves and necrosis often is more severe.

Beta vulgaris L. var. *cicla* L. (Swiss chard).—Symptoms of yellows on Swiss chard are very similar to those produced on sugar beet.

Spinacea oleracea L. (spinach).—Symptoms of yellows on spinach are similar to those found on sugar beet but there appears to be a wider range in severity of reaction to the virus. Under greenhouse

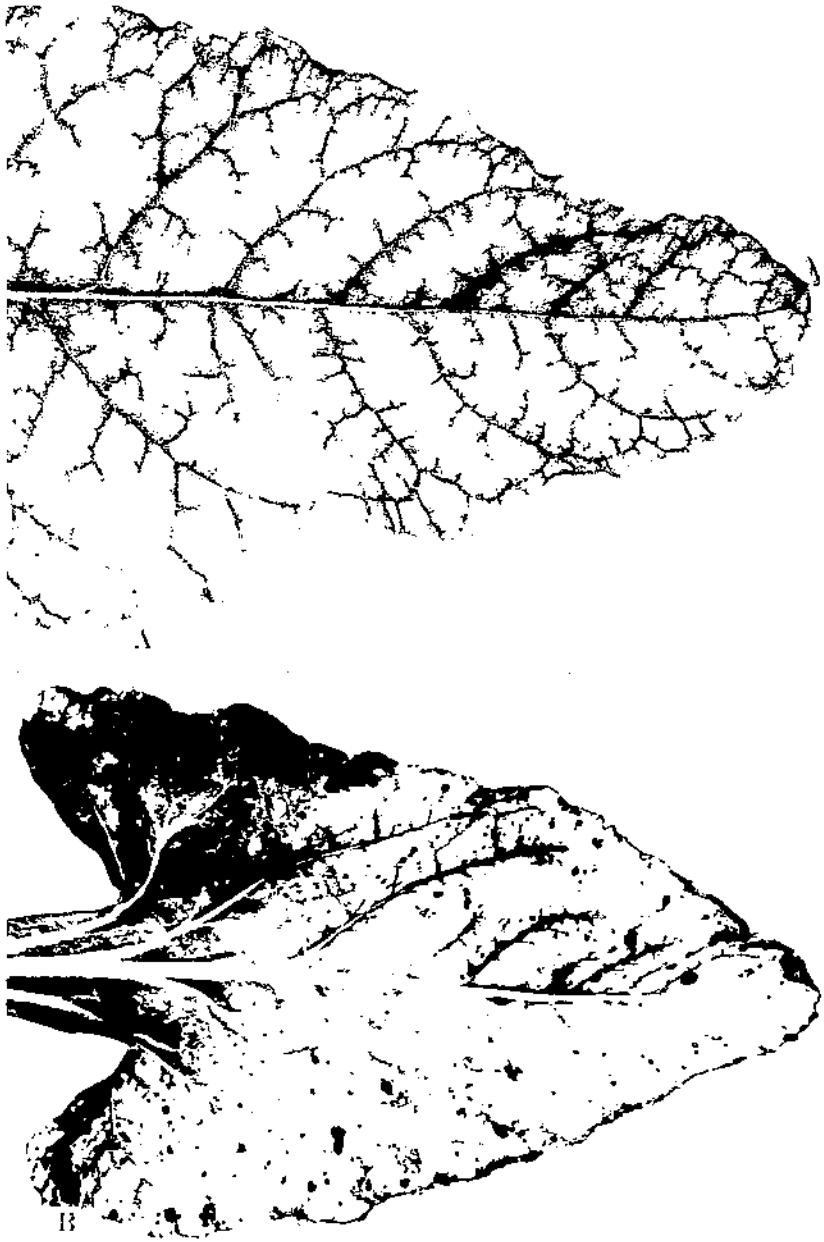

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Figure 4.—*A*, Chronic effects of yellows in which beet leaves thicken, the veins remain green, and the interveinal tissue yellows. *B*, Late stage of yellows development on beet leaf in which tissue between veins becomes necrotic.

conditions, vein clearing may appear in young leaves 7 to 14 days after infection, sometimes accompanied by necrosis. Leaves just older than those showing vein clearing may show various types of chlorotic spotting. Some infected plants die; others survive and produce varying amounts of yellowing on mature leaves.

In the field, yellowing of older leaves is common and usually more intense at the leaf tips and along the margins of the leaves. If symptoms are mild, the older leaves may be more or less mottled with the veins remaining green. The range of yellowing is so great that in some plants it may be inconspicuous, whereas, in others it may be extensive enough to destroy the value of the crop. In some plants the central leaves may show vein clearing and curling soon after infection. In rare cases, the central leaves on young plants may become necrotic. This sometimes leads to death of the plant. Plants may show an appreciable amount of dwarfing and some necrosis even in the absence of marked yellowing (fig. 5)

Symptoms on Weeds and Other Host Plants

The yellows virus produces a variety of reactions on various weed hosts, which may range from no obvious symptoms, as in *Stellaria media* (chickweed), to death of the plant, as in *Beta macrocarpa*. The more virulent strains produce vein clearing in young leaves of some species but not in others. A type of mottling is produced on leaves of *Chenopodium amaranticolor*. Some species recover from certain phases of the disease; others show no evidence of recovery. In general, the virus causes intensification of red color in plants that naturally have red pigment, but red color was partially suppressed in a type of *Amaranthus caudatus* that normally has an abundance of red pigment.

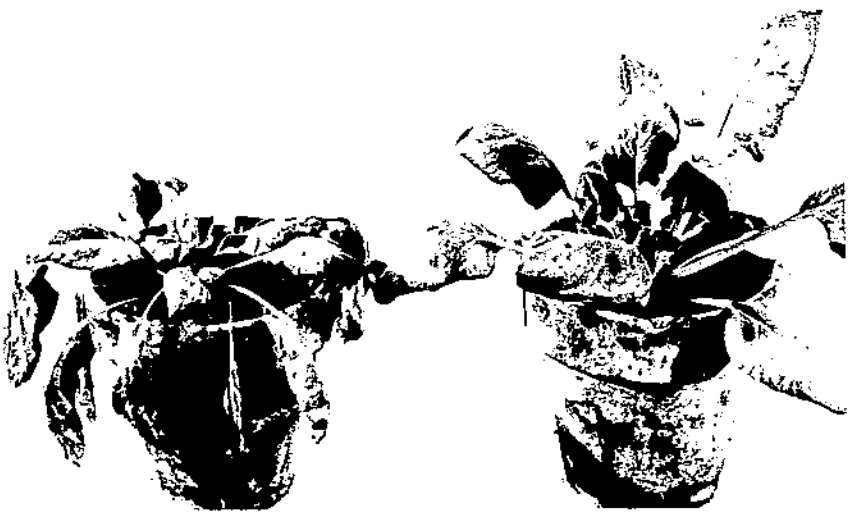

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Figure 5.—Spinach under greenhouse conditions: Left, drooping yellow leaves caused by yellows; and right, healthy check plant.

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The range of effects of yellows is indicated by the descriptions of symptoms on the following species of plants.

Amaranthus retroflexus (redroot amaranth).—This species apparently has a medium degree of susceptibility to infection, and diseased plants are sometimes found in the field. First symptoms usually begin to appear on mature leaves 15 to 20 days after infection and consist of chlorotic splotches in the interveinal areas, followed by yellowing and production of red pigment in and around the chlorotic splotches. In older leaves the interveinal tissue may show some necrosis. Immature leaves show little effects of the disease. Affected plants are not markedly stunted and leaves are nearly normal in size and do not abscise prematurely.

Other species of *Amaranthus* shown to be susceptible produce symptoms similar to those produced on *A. retroflexus*, but injury is less extensive on *A. graecizans*.

Atriplex hortensis (garden orach).—Inconspicuous chlorotic spots are produced on leaves, but there is no leaf deformity. Diseased plants are not dwarfed, but flowering is irregular and delayed 10 days to 2 weeks. Fruits ripen irregularly and the yield of seed is greatly reduced.

Atriplex rosea (redscale).—Plants inoculated when small show symptoms after 20 to 30 days. Mature leaves produce inconspicuous circular, chlorotic spots 2 to 3 mm. in diameter. No vein clearing or necrosis is observed. Although leaf symptoms are very mild, diseased plants are markedly dwarfed and the infected plants at maturity are only about one-fourth to one-half the size of healthy check plants, as illustrated in figure 6.

Atriplex semibaccata (Australian saltbush).—On rapidly growing plants in the greenhouse three or four leaves may show vein clearing that persists in the form of chlorosis of the larger veins. Later growth may show splotching and yellowing between veins, but no vein clearing. Symptoms have not been recognized on infected plants growing under natural conditions.

Beta macrocarpa.—This species is severely injured by the more virulent strains of the yellows virus. First evidence of infection consists of marked vein clearing in the younger leaves, often more evident at the tip, followed by yellowing and thickening of the leaf blades. Leaves die prematurely. Strain 6 (low in virulence) produced no obvious symptoms under green house conditions. However, many plants infected with the more virulent strain 5 were killed, which indicates a very wide range of reaction to strains of different degrees of virulence.

Beta maritima.—This species appears to be relatively resistant to injury by yellows virus, but plants from different seed collections have shown a wide range of yellowing. Symptoms are similar to those produced on sugar beet.

Beta patellaris.—Usually, symptoms begin to appear about 12 days after inoculation and consist of small, circular, chlorotic, spots that appear first on the tips of half-mature leaves and extend to the rest of the leaf as it matures. Chlorotic spots, sometimes more or less associated with the larger veins, appear on leaves of all subsequent growth. There is no tendency toward recovery. No symptoms were observed

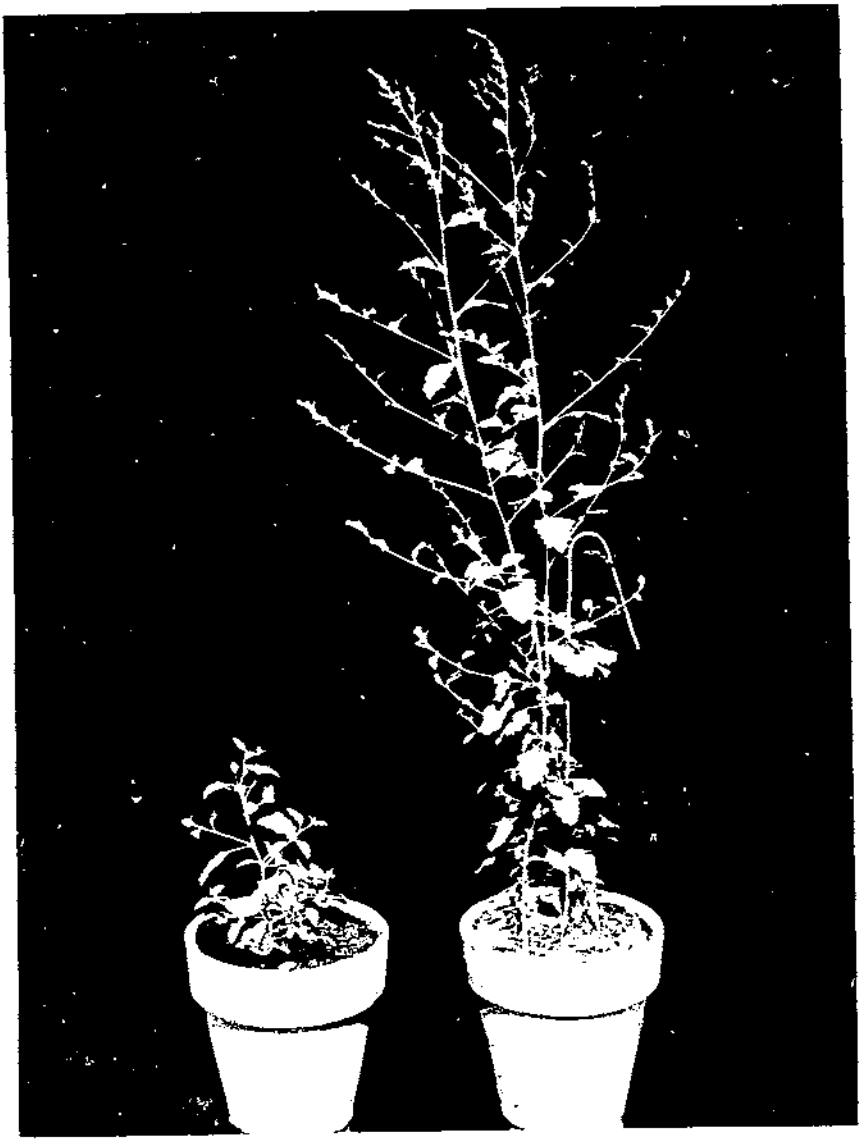

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Figure 6.—Atriplex rosea: Left, dwarfing caused by yellows, as compared with healthy plant (right).

on the young leaves on any infected plants. Very little stunting was produced by the virus strains tested.

Chenopodium amaranticolor.—Obvious symptoms are produced on this species by all the described strains of yellows virus, except strain 6. First symptoms usually consist of a type of indistinct vein clearing accompanied by spotting that approaches a type of mottling. As the plant grows, symptoms continue to be produced on all new growth as the leaves mature. Mature leaves appear mottled as a result of the

production of numerous chlorotic spots, 2 to 3 mm. in diameter, with indefinite margins. The first leaves that become infected may show carmine splotches. Leaves abscise prematurely. Virulent strains cause considerable dwarfing, and dwarfing appears to be more or less proportional to the virulence of the strain of virus involved. Aside from dwarfing, symptoms produced by the different virus strains are similar.

Chenopodium capitatum (strawberry blite).—The edges of young leaves of plants infected with a virulent strain of the yellows virus begin to roll upward 8 to 15 days after infection, petioles may become twisted, and the leaves may show distinct vein clearing. Leaves just older than those showing vein clearing may develop an abundance of red pigment. Young leaves may become necrotic, rate of growth is reduced, plants deteriorate, and many die. Very young plants may be killed rather quickly and may show only vein clearing and dwarfing of young leaves in addition to necrosis. Injury is more or less proportional to virulence of the strain of virus involved. Very mild strains produce little more than reddening of the older leaves.

Chenopodium murale (nettleleaf goosefoot).—Age of the plant appears to influence greatly the susceptibility of this species to infection. Older plants are relatively resistant, but seedlings may be infected readily. Injury by virulent strains of yellows virus is severe on all infected plants. First symptoms begin to appear 7 to 20 days after infection and consist first of twisting of petiole of one or more of the upper leaves, followed soon by vein clearing in the leaf blade. Vein clearing may occur first only on one side of a leaf or in a single sector. Affected leaves tend to roll upward at the edges. Leaves formed later show vein clearing and a small amount of upward curving along the edges, and become slightly thickened and brittle. They eventually yellow and abscise. It is characteristic of this plant species that vein clearing is evident on young leaves throughout the period of growth of the plant and there is little or no tendency toward recovery. Plants infected in the seedling stage are severely stunted and rarely attain more than half the height of healthy plants.

Nicotiana clevelandii.—This species appears to be relatively resistant to infection and not severely injured by the disease. Symptoms produced by virulent strains of the virus are easily recognized and appear first on leaves approaching maturity as marked vein clearing 15 to 20 days after infection. Vein clearing continues to appear on all new growth, and there is no evidence of recovery. Older leaves yellow prematurely and may show necrosis. Symptoms produced by strains of low virulence are restricted to yellowing of older leaves.

This species has a high degree of susceptibility also to other viruses that attack sugar beet. It is susceptible to the viruses of curly top and cucumber mosaic in common with a number of other species of *Nicotiana*. It is susceptible to infection with the viruses of beet mosaic (4) and yellow net; viruses to which most of the other species of *Nicotiana* appear to be immune. The species is known to be an excellent test plant for a large number of viruses that attack plants other than sugar beet.

Plantago erecta.—Symptoms begin with the production of red coloration at the tips of the older leaves of this species about 2 weeks after infection. The red color spreads downward, usually until the entire

leaf is involved, and the leaves die prematurely. Leaves and plants are considerably dwarfed. *P. insularis* produces similar symptoms, except the obviously affected leaves are yellow instead of red.

Salsola kali var. *tenuifolia* (Russian-thistle).—Plants inoculated when about 3 cm. tall begin to show stunting about 20 days after infection. Older leaves show a slight amount of twisting and tend to turn yellow prematurely. After the production of first symptoms, the plants produce new growth that is free of symptoms. Infected plants are dwarfed, however, and at maturity are only about two-thirds the size of healthy check plants.

Senecio vulgaris (common groundsel).—Young leaves begin to show vein clearing about 10 days after infection, and leaves produced later tend to yellow prematurely and to develop red color at the tips and around the leaf margins. Plants are somewhat dwarfed. A few infected plants have been found in beetfields, but the species appears to have considerable resistance to infection under field conditions.

Tetragonia expansa (New Zealand spinach).—Plants inoculated with a virulent strain of virus in the cotyledon stage show distinct vein clearing in the first true leaves. Young leaves continue to show vein clearing as long as normal growth continues (fig. 7, A). Older leaves turn yellow prematurely. Plants are decidedly dwarfed. In later stages of plant growth, sunken, brown, necrotic lesions, up to 1.5 cm. in length and 3 to 4 mm. wide, appear on the stems (fig. 7, B). These lesions result from death of cells to a depth of 1 mm., or less.

Less virulent strains do not produce vein clearing in young leaves. Some plants infected with strains of low virulence show a type of faint mottling, but usually the only evidence of disease consists of premature yellowing of mature leaves. Strains of medium or low virulence have little effect on growth.

ECONOMIC IMPORTANCE OF YELLOWS

Yellows is one of the major diseases of sugar beet in most areas where it occurs, and it is capable of causing severe losses in crops grown for sugar as well as in crops grown for seed. The disease causes severe losses also in table beet, and closely related plants such as Swiss chard, and it is a very destructive disease of spinach under certain conditions.

Damage to the Sugar Beet Root Crop

Extensive damage from yellows has been reported from widely separated areas. Hull (*31*) and Watson and coworkers (*57*) estimated that plants infected in June and July in England lost up to half of their potential sugar yield.

In the Netherlands, Hartsuijker (*27*) found that if infection reaches 100 percent by the end of June losses of 25 to 30 percent of the crops may occur. Results of tests by Björling (*10*) indicate that losses in Sweden may be as high as 61 percent in beets infected early in the season.

Bonnemaison (*12*) estimated that yellows in France may reduce root yields 30 to 40 percent.

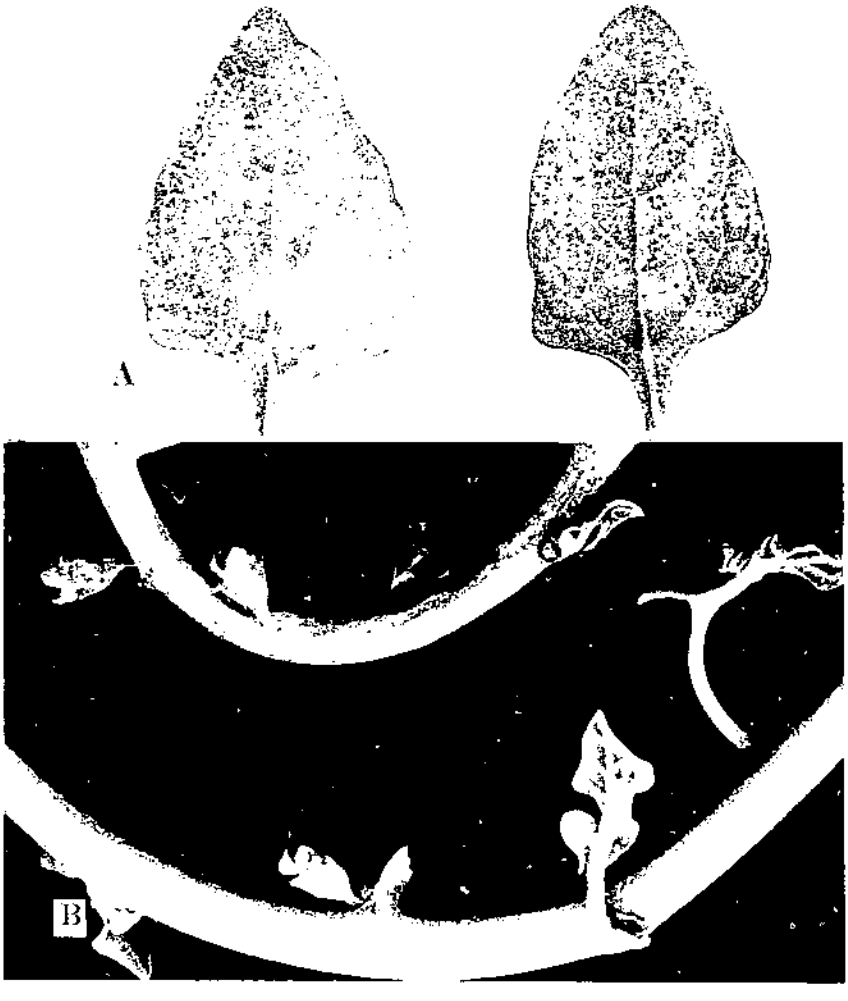

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Figure 7.—New Zealand spinach: *A*, Leaves showing vein clearing, which symptom continues to be produced on new growth in chronically infected plants; *B*, stem (upper) showing depressed necrotic lesions produced on plants infected with virulent strains of yellow virus, and stem (bottom) from healthy check plant.

Kovács (*53*) estimated that 50.4 percent of the sugar beet seed crop, 22.4 percent of the beet root crop, and 25.2 percent of the forage beet crop were infected in Hungary in 1953. Losses in sugar yields up to 35.2 percent were reported in controlled experiments.

Wiesner (*59*) found losses in root weight in tests in Germany ranged from 35.1 to 42.1 percent in tests in 1956 and from 12.5 to 54.6 percent in tests in 1957. Reduction in sucrose percent in the 2 years ranged from 1.1 to 2.1 percentage points.

In the United States, losses are limited largely to those areas where sugar beet, or other susceptible crop plants, are present during the entire year. The seriousness of the disease depends on the age of the plants when infected, the virulence of the strains of virus involved, and possibly the conditions under which the crop is produced.

In tests in Colorado in 1953, Coons, Gaskill, and Daniels (*18*) found that beets inoculated July 16 yielded 6.8 percent less than controls in which considerable natural infection occurred. They estimated that if comparisons had been made on the basis of 100 percent infection versus 100 percent healthy plants, yields would have shown a depression in root growth of 10 to 15 percent. Apparently, infection in Colorado usually occurs later in the development of the plant than in some other areas and losses are correspondingly less serious. This appears to be true in the sugar-beet-producing areas of Washington and Oregon.

In the Imperial Valley of California, where plantings are made in August and September, symptoms began to appear in March in 1957 and a high percentage of infection was evident in a number of fields by April 15, which was the beginning of the harvest period in that area. A similar condition was noted in 1958 and 1959. Late infection of this type would not be expected to cause serious losses by the beginning of harvest, but there is some evidence that beets with yellows were more seriously damaged by root rot and other unfavorable conditions in June and July as temperatures increased toward the end of the harvest period. The direct and indirect damage caused by yellows under such conditions is difficult to estimate.

In the San Joaquin Valley of California, a large share of the infection that occurs takes place in March and April and sometimes extends into early May. The amount of infection has varied from year to year, but it was unusually extensive in 1957 and 1958. Yields and sugar content were very low in many fields in both years. Yellows was undoubtedly a factor in these reduced yields, but its importance in relation to other conditions has not been determined. San Joaquin Valley is an area with high summer temperatures, and it is possible that yellows may increase the damage caused by other factors, such as very high temperatures and curly top.

Greatest direct losses to the beet crop probably occur in central California, where many fields show high percentages of infection by June 1. Results of replicated tests in the Salinas Valley in central California and at Riverside in southern California, which extended over a period of 5 years, have been reported and reviewed by Bennett, Price, and McFarlane (*8*). These results, summarized in table 1, indicate that yellows is capable of causing heavy losses. Reduction in root yields ranged from 2.0 to 47.0 percent and reduction in sucrose content ranged from 0.1 to 3.1 percentage points.

Reduction in root weight was markedly influenced by the virulence of the strain of virus involved. Strains of high virulence caused losses up to nearly twice those caused by strains of low virulence. Reduction in sucrose percentage also appeared to be correlated, to some degree at least, with virulence of strain of virus used.

One of the most important factors involved in losses caused by yellows is age of plant at the time of infection. Inoculation of plants in the 12- to 16-leaf stage in tests at Salinas, Calif., in 1955 resulted

TABLE 1.—*Summary of results of tests made in California to determine the effect of yellows on tonnage and sucrose content of sugar beet*

Year of test	Location of test [1]	Virus used	Average yield of beets		Average sucrose	
			Yellows infected	Check	Yellows infected	Check
			Tons per acre	*Tons per acre*	*Percent*	*Percent*
1952	Salinas	Field	20. 4	20. 8	16. 6	19. 7
1952	do	do	22. 3	34. 4	15. 3	16. 4
1953	Riverside	Strain 2.	18. 7	28. 8	15. 2	15. 3
1954	do	do	24. 4	38. 8	10. 5	12. 0
1955	do	do	24. 6	35. 2	16. 0	17. 4
1955	do	Strain 6.	29. 2	35. 2	16. 2	17. 4
1955	Salinas	Strain 5.	17. 1	26. 1	15. 3	17. 7
1955	do	Field	19. 6	25. 6	14. 1	15. 5
1956	do	Strain 3	22. 6	30. 4	16. 1	17. 2
1956	do	Strain 5	16. 1	30. 4	15. 2	17. 2

[1] Tests at Salinas were made in cooperation with J. S. McFarlane; those at Riverside in cooperation with Charles Price.

in a reduction of 34.1 percent in root weight, whereas inoculations 49 days later resulted in a reduction in root weight of only 12.5 percent. Similar results were obtained in further tests in 1956 (*8*) in which inoculations with a virulent strain of virus March 25, April 30, and June 5 resulted in reduction in root yield of 47.0, 33.9, and 13.5 percent, respectively.

Variation in the importance of different factors involved in losses caused by the yellows disease in different areas where yellows is prevalent makes estimates of the damage to the beet crop, as a whole, difficult and unreliable. In plot tests in the Salinas Valley in 1955, natural infection caused a reduction in tonnage of 22.3 percent and a reduction in sucrose of 1.38 percentage points. As there was some damage to the check plots in this test, it seems probable that losses were somewhat higher. On the basis of this test and other information, it seems probable that if fields are heavily infected by the early part of June, tonnage losses may be 25 percent or greater. If infection occurs later, losses are correspondingly lower.

Damage to the Sugar Beet Seed Crop

Yellows may cause severe injury to the sugar beet seed crop, but losses undoubtedly vary considerably under different conditions. In experimental tests in England, Hull (*30*) reported that the effects of the disease on seed yield depended on the date of infection of stecklings, but plants infected in early stages of development yielded only 40 to 60 percent as much as healthy plants. Bonnemaison (*12*) estimated that yellows may reduce seed yields 40 to 50 percent in France.

Striking reductions in yield of seeds of sugar beet and fodder beets due to mixtures of yellows and mosaic viruses have been reported by Schlösser in Germany (*47*). Time of infection greatly influenced re-

duction in yield. In tests at Einbeck, yields of seed from plants infected in the summer of the first year, late autumn of the first year, early summer of the second year, and from healthy plants were 10.5, 26, 82, and 121 grams per plant, respectively. More or less comparable losses were found in fodder beets with this virus combination. The effects of yellows alone were not determined.

Other tests in Germany by Wiesner (*60*) indicated that infection of beets with a combination of beet mosaic virus and yellows virus in the steckling stage reduced seed yields 45 to 65 percent and infection in the early stages of seedstalk production reduced yields 40 to 50 percent. Seed quality was influenced less than seed yield. The effects of the combination appeared to be a summation of effects of the two viruses separately.

Damage to the sugar beet seed crop in the United States varies with the area in which the crop is grown. Sugar beet seeds are produced in Salt River Valley of Arizona, western Oregon, southern Utah, and in the Hemet Valley and the Tehachapi area of California. Yellows undoubtedly causes damage in the two principal seed-producing areas, namely, western Oregon and the Salt River Valley. Some fields in Oregon have shown high percentages of infection in the spring. Yields were depressed in some fields in 1957 and 1958, but the importance of yellows in these reduced yields was not determined.

The yellows disease was observed in the Salt River Valley first in 1955. Infection probably ranged from 20 to 100 percent in 1955 and in succeeding years through 1958, but much of the infection occurred after bolting began. Tests were made by Hills and others (*29*) in 1957-58 to determine the effects of yellows on yield and quality of seed. Field plots were inoculated November 20, 1957, and February 21, March 26, April 6, and April 21, 1958. Reductions in yields were 1,495, 1,176, 767, 587, and 310 pounds per acre, or 35.0, 27.5, 17.9, 13.7, and 7.2 percent in plots inoculated on the respective dates. Even the last date of inoculation, made April 21 when the plants were in blossom, produced a 7.2 percent reduction in yield and an 8.4 percent reduction in germination of seeds. Seed germination was lower also in plots inoculated February 21, but inoculation on other dates apparently did not affect germination. Weight of individual seeds was lower in all dates of inoculation.

Four tests were made in the greenhouse, in which an annual type of beet from the Imperial Valley of California and three selections of sugar beet were used, in an attempt to obtain further information on the effects of yellows on seed production. Seedlings were planted in pots, inoculated with yellows virus, either about the six-leaf stage or shortly after they had started to bolt, and carried to seed production under greenhouse conditions. The three selections of sugar beet were subjected to periods of low temperature sufficiently prolonged to induce bolting.

The results of these tests are shown in table 2. Yellows markedly decreased seed yield in the annual type of beet and in S.L. 54484+0. Less reduction in yield was produced in U.S. 33, and there is no evidence of yield reduction in U.S. 22 under conditions of these tests. Reduction in weight of individual seeds closely paralleled total weight reduction. There is no evidence that yellows appreciably affected seed germination. Absence of an effect on seed germination has been indi-

TABLE 2.—*Effect of yellows on beet seed production under greenhouse conditions*

Selection tested and stage of plant development at time of inoculation	Plants that fruited	Bolting	Average weight of seeds per plant	Average weight of 100 seeds	Germination
	Number	*Percent*	*Grams*	*Grams*	*Percent*
Imperial annual beet:					
6-leaf stage	27	54	0. 57	1. 021	27. 6
Early bolting	48	96	. 81	1. 225	25. 6
Noninoculated	50	100	1. 55	2. 487	29. 6
S.L. 54484+0 sugar beet:					
6-leaf stage	44	73	6. 44	1. 963	76. 8
Early bolting	46	77	6. 72	1. 503	77. 5
Noninoculated	49	82	10. 12	2. 079	74. 0
U.S. 33 sugar beet:					
6-leaf stage	72	72	5. 35	1. 705	93. 3
Noninoculated	96	96	6. 36	1. 721	80. 2
U.S. 22 sugar beet:					
6-leaf stage	32	52	8. 62	2. 031	85. 0
Early bolting	42	70	8. 42	2. 442	77. 7
Noninoculated	44	73	8. 30	2. 208	74. 3

cated also in other tests in which seeds were harvested from individual diseased and from symptomless plants and tested for germination. Seeds of diseased plants of commercial varieties tested in the Salinas Valley have germinated in excess of 85 percent in almost all instances, and it appears that under these conditions yellows has not obviously depressed viability of seeds.

In the greenhouse tests, and also under field conditions, bolting is delayed in plants infected in early stages of development. This delay usually averages 7 to 10 days. In some varieties, yellows probably reduces the percentage of plants that produce seeds, particularly under conditions in which plants have been subjected to thermal induction inadequate for the production of a high percentage of bolting in healthy plants.

In tests at Salinas, Calif., in 1956–57 and 1957–58, yellows produced reduction in yield ranging from 21.1 to 70.2 percent (*7*). The amount of damage was influenced to a high degree by the time the plants were infected. Weight of individual seeds was lower in plots inoculated in March than in plots inoculated in May and appreciably lower than weight of seeds from the check plots. There was no evidence that yellows depressed germination. Preliminary evidence indicated that varieties and selections may differ appreciably in their susceptibility to injury by yellows.

Damage to the Table Beet and Spinach

Very little information is available on the effects of yellows on yield and quality of the table beet. Infected plants, however, are obviously dwarfed in some fields. McLean (*37*) reported that in Washington, seed plants of table beet with yellows produced smaller seeds and seeds of lower vitality than virus-free plants.

Yellows has proved to be a serious disease of spinach in the Salinas Valley of California. The damage varies and probably is influenced greatly by environmental conditions. Some fields with high percentages of infection show little yellowing and little reduction in quality of spinach, but yields may be reduced appreciably. Other fields have shown so much yellowing of older leaves that they have not been harvested.

Secondary Effects of Yellows

In addition to the direct injury caused by yellows, the yellows disease probably augments the injury caused by certain other diseases. Giddings (*25*) reported that under greenhouse conditions beet plants with yellows were more susceptible to infection with curly top virus and more severely injured by the curly top disease than yellows-free check plants. A similar effect was noted in field tests at Riverside, Calif., in 1955 in variety tests in which alternate blocks of 200 selections were inoculated with yellows virus. Considerable natural infection with curly top virus occurred, which permitted observations of the effects of curly top and yellows, alone and in combination, on plants of each selection. In many instances plants with yellows were more severely dwarfed by curly top than were yellows-free plants. The effects of the combination were rather spectacular on certain selections, particularly on some of the selections that had considerable natural resistance to curly top. Plants of some of these selections that were damaged very little when affected with curly top alone were very severely dwarfed by the combination of curly top and yellows.

In fields in California in which yellows has occurred in combination with leaf spot, caused by *Cercospora beticola* (Sacc.), it has appeared that plants infected with yellows virus were much more severely affected by leaf spot. Kovács (*33*) reported that in Hungary, yellows-diseased plants showed increased susceptibility to leaf spot. Schlösser (*40*), in Germany, states that the resistance to leaf spot was largely overcome by inoculating with yellows virus in mid-June and that the losses caused by leaf spot were considerably greater on plants with yellows than on plants that were free of yellows infection. Heiling and others (*28*) reported increased leaf spot susceptibility of yellows-infected plants in field tests in Germany and found the *C. beticola* grew better on media prepared from yellows-affected leaves than on media prepared from healthy leaves.

The effects of yellows in combination with beet mosaic were studied by Lüdecke and Neeb (*36*) in Germany. They found that, in general, the losses resulting from mixed infection approximated the sum total of the quantitative and qualitative depreciations due to both viruses separately.

The number of wilting and dying plants found in a number of spinach fields in the Salinas Valley of California indicates that yellows in spinach may render spinach plants much more susceptible to certain types of vascular parasites and root rot organisms.

Yellows therefore may be expected to increase the damage caused by cercospora leaf spot and curly top. It may be that losses caused by rhizoctonia root rot and certain other soil-borne organisms are

also increased by infection with the yellows virus. In some instances it may be that secondary damage of this type may be more severe than that caused by direct effects of yellows.

TRANSMISSION OF THE YELLOWS VIRUS

Transmission by Insects

Several species of aphids have been shown to be vectors of the sugar beet yellows virus. Van Schreven (*51*) reported transmission of the virus by the bean aphid, *Aphis fabae*, in 1936, and Roland (*44*) reported transmission by both *A. fabae* and the green peach aphid, *Myzus persicae* (Sulz.), the same year.

Doncaster and Kassanis (*20*) found that the shallot aphid, *Myzus ascalonicus* Doncaster, is able to transmit the yellows virus, but they considered it of little importance as a vector.

Hartsuijker (*27*) reported transmission of yellows virus by the aphids, *Rhopalosiphoninus tulipaellus* (Theob.), *R. latisiphon* Davidson, *Doralis rhamni* B. d. F., *Macrosiphum solanifolii* (Ashm.), and *Myzus solani* (Kalt.) (*Aulacorthum solani* Kalt.). In the Netherlands *R. tulipaellus* moved from mangel clamps to beetfields in the spring, and for this reason this aphid was considered important in the initial spring spread of the virus to beetfields.

Of the species of aphids known to be vectors, *Myzus persicae* and *Aphis fabae* appear to be the most important in the spread of the yellows virus in Europe. Watson and coworkers (*58*) state that, in England, spread of yellows is more closely correlated with populations of *M. persicae* than with populations of *A. fabae*. Rietberg (*42*) states that, in the Netherlands, field studies of aphid populations have indicated that in some years *A. fabae* plays the major role in yellows virus spread, whereas in other years *M. persicae* is the more important vector.

Szirmai (*53*) states that the main vector in Hungary is *Aphis fabae* and that *Myzus persicae* is rarely observed there on sugar beet.

In 1952 tests were begun in the United States to obtain evidence on the ability of various species of aphids to transmit the yellows virus. Colonies of aphid readily available were established on appropriate host plants. Each species of aphid was either obtained from a known authentic source or collected from the field and submitted to an entomologist for identification. Aphids were transferred from infected sugar beet plants, or other species of plants infected with yellows virus, to healthy seedling plants. Most tests were made with strains of the yellows virus that produce marked vein clearing on the younger leaves of sugar beet plants.

The results of these tests, shown in table 3, indicate that 7 of the 14 species of aphids tested are able to transmit the yellows virus. The tests indicate a rather wide range of efficiency in transmission among the different vectors. The green peach aphid, *Myzus persicae*, appears from these and other tests to be a very efficient agent of transmission. The high efficiency of *Aphis cuscutae* Davis as a vector is somewhat surprising in view of the fact that it lived only 2 to 3 days on the plants on which it was tested. Some of the species

TABLE 3.—*Results of tests of species of aphids for ability to transmit sugar beet yellows virus*

Species of aphids tested	Plants inoculated	Plants infected
	Number	*Number*
Aphis cusculae	57	33
A. fabae	40	13
A. gossypii	76	15
A. medicaginis	184	0
Hyalopterus atriplicis	74	0
Hysteroneura setariae	34	0
Macrosiphum chrysanthemi	46	0
M. pisi	107	8
M. solanifolii	40	11
Myzus hieracii	68	0
M. ornatus	12	4
M. persicae	60	54
Pemphigus betae	113	0
Rhopalosiphum maidis	40	0

that did not transmit the virus were given extensive tests. *Pemphigus betae* Doane, among the nonvectors, occurs abundantly on roots of beet and other plants in areas where yellows is prevalent. This aphid does not feed readily on green tops of beet plants, but it will feed and multiply on etiolated beet leaves in the dark. Therefore, first tests were made by transferring aphids from the roots of yellows-infected plants to etiolated leaves of healthy plants in the dark. None of the inoculated plants became infected.

In other tests with *P. betae*, beet seeds were allowed to germinate in the dark, and aphids from the roots of diseased plants were placed on the young seedlings. The aphids fed readily on both tops and roots of the seedlings under these conditions. After feeding periods of 24 to 72 hours, the aphids were destroyed and the inoculated seedlings were transferred to pots in the greenhouse. Since no infection occurred under these conditions, it is assumed that this species is not a vector of the yellows virus. This conclusion is supported by the fact that there has been no evidence of spread of yellows virus from yellows-infected plants in greenhouses used for yellows tests over a period of 7 years under conditions of light to heavy infestation of roots of both healthy and diseased plants by this aphid.

Aphis medicaginis Koch was tested on both sugar beet and *Atriplex rosea*. The latter plant is a host of *Aphis medicaginis* and of the yellows virus that permitted the breeding of the aphid on diseased plants and transferring them in large numbers to healthy seedlings. *Macrosiphum chrysanthemi* (Oest.) was tested in a similar manner on *Senecio vulgaris*. As no infection was obtained, it seems highly probable that neither of these aphid species is a vector of yellows virus.

Transmission by Juice Inoculation

Transmission of the sugar beet yellows virus by juice inoculation was obtained first by Kassanis (*32*) in 1949, who reported that small numbers of inoculated plants developed local lesions on rubbed leaves

and that some of the plants that showed local lesions later developed systemic infection. Watson (*56*), using techniques similar to those employed by Kassanis, reported good results in cross-protection tests with strains of yellows virus.

Coons (*16*), using yellows virus in the United States, reported systemic infection in a relatively high percentage of inoculated plants, but he observed no local lesions. Costa and Bennett (*19*) transmitted yellows virus to sugar beet and to *Chenopodium murale* by juice inoculation and obtained both local lesions and systemic infection. Production of infection, however, was very erratic, and only a relatively small percentage of the inoculated plants showed symptoms. Usually, but not always, production of local lesions was followed by systemic infection.

More recently, Mundry and Rohmer (*38*) reported mechanical transmission of yellows virus to sugar beet and *Chenopodium foliosum* (Moench.) Aschers in Germany.

Beginning in 1955, a more extensive search in the United States was started in an effort to find a plant more susceptible to infection by juice inoculation than the sugar beet varieties that had been used. Tests were made of more than 300 selections of sugar beet, but none of these appeared to be more susceptible than the variety U.S. 75 used in earlier experiments. Tests were made also of most species of plants that had been shown by inoculation with aphids to be susceptible to infection with the yellows virus. Most of these species appeared to be immune from or very resistant to infection by the rubbing method of inoculation. Some local lesions were produced on leaves of *Chenopodium murale* and spinach, but inoculated plants rarely showed systemic infection. A few local lesions were found on inoculated leaves of *C. amaranticolor*, but none of the inoculated plants became infected systemically.

Of the species of plants tested, *Chenopodium capitatum* gave the highest number of local lesions. In fact, under certain conditions, this plant is a very excellent local-lesion host of the yellows virus. Lesions often are produced in large numbers, and they are sufficiently distinct to be counted readily. Plants of this species were used extensively in further studies of conditions favorable for infection and for studies of properties of the yellows virus.

Lesions on Chenopodium capitatum

Local lesions may begin to appear on leaves of plants of *Chenopodium capitatum* 5 days after inoculation, but often longer periods are required. After the first lesions are visible, others may continue to appear for several days, which indicates a variation in rate of cell invasion by the virus. Lesions are circular and rarely attain a diameter of more than 1 mm. They first appear as slightly sunken, watersoaked spots, visible only by reflected light and only on the inoculated side of the leaf. Within 2 or 3 days after they become evident, the lesions become visible on both leaf surfaces as light-colored, slightly sunken, necrotic spots that tend to fade to almost white with age. A leaf of *C. capitatum* with typical local lesions as they appear 10 to 15 days after inoculation is shown in figure 8, *B*. Lesions on sugar beet and *C. murale* are shown in figure 8, *A*, and 8, *C*, respectively.

The first evidence of injury to individual cells by the virus, as indicated in freehand sections of infected leaves of *C. capitatum*, is the

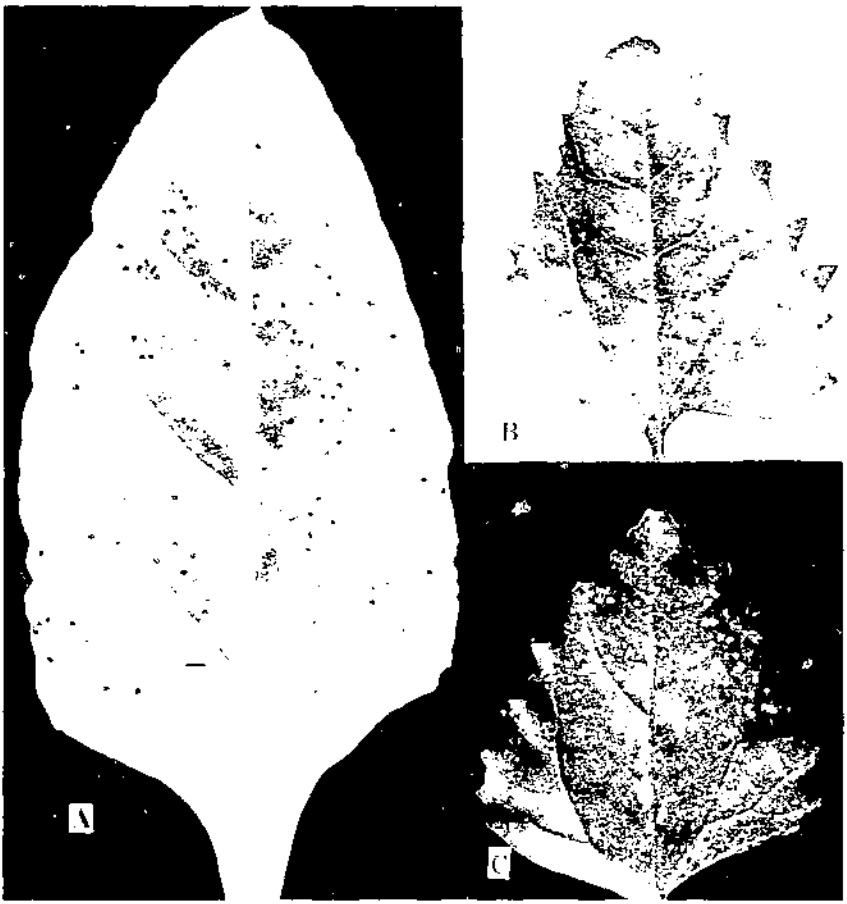

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Figure 8.—Leaves showing local lesions produced following juice inoculation of (*A*) sugar beet, (*B*) *Chenopodium capitatum*, and (*C*) *C. murale*.

shrinking and darkening of the protoplasm in epidermal, palisade, and mesophyll cells in the invaded area. Protoplasm disintegration progresses rapidly, cell walls become discolored, and some walls partially collapse, resulting in shrinking in the upper part of the lesion, which includes the epidermis. Spread in depth usually is more rapid than lateral spread, probably owing to more rapid movement of virus in the elongated palisade cells.

Spread of the virus in parenchyma appeared to stop with collapse of a certain number of invaded cells. Many plants on which local lesions were produced did not become infected systemically. The number of local lesions produced was closely correlated with the probability of systemic infection (table 1). Only 10.2 percent of the plants with 25 lesions or less became infected systemically. Percentage of systemic infection increased with increase in number of local lesions, but 100 percent systemic infection was observed only after the

TABLE 4.—*Relation of number of local lesions per plant to production of systemic infection following mechanical inoculation of Chenopodium capitatum*

Local lesions per plant (number)	Plants inoculated	Plants infected systemically	
	Number	*Number*	*Percent*
25 or less	176	18	10.2
26 to 75	86	33	38.4
76 to 125	49	33	67.3
12 to 200	18	15	83.3
201 to 300	18	18	100.0
301 or more	7	7	100.0

number of local lesions reached 200 per plant. These results indicate that virus introduced into the parenchyma by the rubbing method of inoculation is limited to parenchyma cells in the majority of lesions but that it may escape into the phloem in a low percentage of instances and produce systemic infection.

Factors Influencing Transmission of Yellows Virus to Chenopodium capitatum

A high percentage of infection of plants of *Chenopodium capitatum* by juice inoculation was obtained only during the winter and early spring. Inoculations during the summer and fall months gave local lesions in some instances, but usually the number was small. The reasons for this seasonal result have not been determined.

The highest numbers of local lesions were produced on plants in the 6- to 8-leaf stage. Larger plants appear to be resistant. The number of local lesions on different leaves of the same plant may range from few or none to 200 or more. Tests made to determine which leaves are most susceptible to infection indicated that with plants in the 8-leaf stage the largest leaf, which on this type of plant is just reaching maturity, usually showed the greatest number of local lesions. Few lesions were produced on the immature leaves or on the oldest leaves. Inoculation of the upper surface of the leaves gave more lesions than inoculation of the lower surface.

The conditions to which plants are subjected before and after inoculation influenced the number of lesions produced on inoculated leaves. Preconditioning in darkness or reduced light, as found by Kassanis (*32*) with sugar beet, resulted in increased numbers of lesions on leaves of *C. capitatum*. However, preconditioning is not always necessary for infection, as indicated by results of tests shown in table 5. Two days in subdued light appeared to be near the optimum period for preconditioning. Shorter periods were not sufficient for proper conditioning, and longer periods caused changes that were likely to result in death of inoculated leaves before lesions had time to appear.

Greater numbers of lesions were obtained on plants that remained in subdued light short periods after inoculation than on leaves that were returned to full light of the greenhouse immediately after inoculation (table 5).

Other tests indicated that washing leaves with tapwater immediately after inoculation caused a reduction in the number of lesions,

TABLE 5.—*Effect of different treatments, before and after inoculation, on production of local lesions on leaves of Chenopodium capitatum*

Time plants kept in subdued light following inoculation (hours)	Lesions on leaves of plants in—			
	Subdued light for 48 hours prior to inoculation [1]		Normal light of greenhouse prior to inoculation [1]	
	Test 1	Test 2	Test 1	Test 2
	Number	*Number*	*Number*	*Number*
0	10.0	9.3	3.0	2.7
2	21.7	23.0	5.7	5.7
4	30.7	37.7	7.7	9.3
6	31.0	31.7	18.0	13.7
8	34.7	32.3	15.0	12.0
24	47.7	41.3	12.3	8.0

[1] Average of 3 leaves.

ranging from 11 to 100 percent. The reduction in number of lesions from washing was greater on plants that were returned to the greenhouse immediately after inoculation than on those that were held in subdued light.

The source of inoculum greatly influences the number of lesions obtained. Plants that have given best results as sources are *Nicotiana clevelandii* and *Chenopodium murale*. Juice from New Zealand spinach has given reasonably high lesion counts in a number of tests, but beets in a chronic stage of disease have been rather uniformly poor as a source of inoculation. In one test that is more or less representative of results of many series of inoculations, the average number of local lesions produced on leaves of *C. capitatum* plants showing the highest count, were 89, 44, 24, and 6 with juice from *N. clevelandii*, *C. murale*, New Zealand spinach, and sugar beet, respectively. In this test juice was pressed from plants that were chronically infected. Juice from beet plants showing incipient symptoms has given higher virus titers than juice from chronically infected plants.

The strain of virus used in making mechanical inoculations is highly important. No lesions and no systemic infections were obtained from inoculations with any one of the three virus strains of low virulence that were used. Each of three vein-clearing strains of the virus used produced local lesions on inoculated leaves, and systemic infection followed in a number of plants. An isolate, designated strain 5, however, consistently gave the highest number of lesions.

Tests for Seed Transmission

Seed transmission of the yellows virus that occurs generally throughout Europe has not been considered of practical significance. Tests in England and the Netherlands have given no seed transmission: however, Nikolić (*39*) in Yugoslavia reported transmission through 4 of 2,064 seedlings, which may indicate that the virus is transmitted through a very low percentage of seeds under some conditions.

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Clinch and Loughnane (*15*) found that a virus that caused symptoms on beets similar to those caused by yellows virus is transmitted through a high percentage of the seeds of a family (No. 41) of sugar beets bred in Eire in 1945. Watson (*56*) states that the Irish yellows virus produces slightly different symptoms, and her evidence from serological comparisons and cross-protection tests on beets indicated that the seed-transmitted virus in Eire is unrelated to the true yellows virus.

Field observations in the United States have not indicated that seedling sugar beets have yellows. Seeds known to be derived from infected plants have given virus-free fields when planted in areas where yellows does not occur.

Greenhouse tests were started in 1953 to obtain additional evidence on the possibility of seed transmission of the virus. Seeds were harvested from available species of plants that showed marked symptoms and that did not recover from symptoms. It seemed reasonable to assume that if the virus is transmissible through the seeds of such plants, symptoms would appear in the seedlings.

The results of these tests, shown in table 6, give no indication that the yellows virus is transmitted through the seeds of any of the species of plants tested. From these results and from observations, it seems unlikely that seed transmission is a factor in the dissemination of virus yellows in the United States. Also, as yet, there is no evidence that the seed-transmitted disease of sugar beets found in Eire occurs in this country.

TABLE 6. *Results of tests to determine whether the yellows virus is transmitted through seeds of diseased plants*

Plant tested	Seedlings from seeds of infected plants	Seedlings diseased
	Number	*Number*
Amaranthus retroflexus (redroot amaranth)	524	0
Amaranthus sp.	701	0
Atriplex rosea (redscale)	342	0
Beta patellaris	163	0
B. vulgaris (sugar beet)	2, 200	0
Celosia sp.	13	0
Chenopodium album (lambsquarters)	890	0
C. amaranticolor	758	0
C. murale (sowbane)	364	0
C. urbicum (goosefoot)	573	0
Nicotiana clevelandii	716	0
Tetragonia expansa (New Zealand spinach)	250	0

Tests With Dodder

The yellows virus is of the general type with which considerable success has been obtained in transmission with different species of *Cuscuta*. However, conflicting results with this virus have been obtained by different investigators.

Fuchs and Beiss (*23*) reported in one test that all 8 plants inoculated with yellows virus in Germany by means of *Cuscuta gronovii* Willd. developed symptoms of yellows after 8 weeks; in further tests, Beiss (*1*) reported a high percentage of transmission with this species of dodder. Canova (*14*), in Italy, failed to transmit two forms of yellows virus by means of *Cuscuta epithymum* Murr., but he found that aphids (*Myzus persicae*) acquired virus by feeding on dodder growing on infected beet plants. This study would indicate that dodder picked up the virus from the diseased plants, although dodder did not transmit the virus to healthy plants.

Rather extensive attempts were made at Salinas, Calif., to transmit the yellows virus by means of *Cuscuta americana* L., *C. californica* Choisy, *C. campestris* Yuncker, and *C. gronovii* Willd. from sugar beet to sugar beet and other susceptible species. Vein-clearing strains of the virus were used in all tests to insure clearly defined early symptoms. The tests were made by establishing virus-free dodder on diseased beet plants and by allowing vigorous stems to grow to adjacent healthy plants. Contacts by dodder between diseased and healthy plants were maintained for at least 7 days in most instances. The results shown in table 7 indicate that none of these species of dodder transmitted the yellows virus in this series of tests.

In further experiments, green peach aphids were allowed to feed on colonies of *C. californica* and *C. campestris* growing on diseased beet plants, and then transferred to seedling beet plants. High percentages of infection were obtained from both species of dodder, showing conclusively that the dodder was able to acquire yellows virus from the infected beet plants. Dodder from the diseased beet plants was allowed to grow to plants of Turkish tobacco. Thirty days after contact with the diseased beet plants was broken, the dodder on the Turkish tobacco plants was tested for presence of the yellows virus by means of aphids. No infection was obtained in these tests, indicating that the virus did not persist in dodder for as long as 30 days after it becomes established on a plant immune to yellows.

TABLE 7. *Results of tests to determine if a vein-clearing strain of yellows virus is transmissible by dodder*

Species of dodder used	Sugar beet plants inoculated	Sugar beet plants infected
	Number	*Number*
Cuscuta americana	110	0
C. californica	150	0
C. campestris	111	0
C. gronovii	82	0

PROPERTIES OF THE YELLOWS VIRUS

Although Kassanis' (*32*) reported juice transmission of the yellows virus on sugar beet in 1949, infection has been so erratic and uncertain with the methods employed that apparently little effort has been made to take advantage of mechanical transmission to determine the

properties of the yellows virus. Tests were made by A. S. Costa and the writer in 1953 to determine the thermal inactivation point of the yellows virus. Good results were obtained in two tests in which sugar beet plants were inoculated with juice from diseased beet plants. These tests indicated that the virus is inactivated by a 10-minute treatment at a temperature between 50° and 55° C. Other tests to determine properties were inconclusive, because of the low incidence of transmission.

Bereks and Zimmer (*9*), using serological procedures, obtained evidence indicating that the thermal inactivation point of the yellows virus lies between 50° and 54° C. (mean 52.5°). The virus retained serological activity in extracted juice for 20 days at 30° and for 4 to 5 days at room temperature.

After it was determined that under proper conditions *Chenopodium capitatum* is highly susceptible to infection when inoculated by means of juice and that large numbers of local lesions are produced on inoculated leaves, further tests were conducted by direct inoculation procedures to learn more of the properties of the virus, particularly its thermal inactivation point, tolerance of dilution, and longevity in vitro.

Thermal Inactivation Point

In tests to determine the thermal inactivation point of the yellows virus, 2-cc. quantities of juice from diseased plants were placed in small, thin-walled tubes and subjected to the desired temperature for 10 minutes. The juice was then used to inoculate leaves of plants of *Chenopodium capitatum*. The results of three tests, selected as representative, are shown in table 8. These results indicate that the thermal inactivation point of the yellows virus lies between 50° and 55° C. However, there was appreciable inactivation at 45° and a high percentage of the virus was destroyed at 50°. This was indicated both by reduction in number of local lesions produced and by the number of inoculated plants that became infected systemically.

Tolerance of Dilution

Extracted juice from infected plants was diluted with tapwater and inoculated into leaves of plants of *Chenopodium capitatum*. The results of three tests, selected as representative, show that the virus survived a dilution of 1 to 500 in one test and 1 to 5,000 in two tests (table 8). Infection dropped sharply as dilution was increased. As would be expected, virus in juice with the higher virus titers showed the greatest tolerance of the higher dilutions.

Longevity in Vitro

Juice was extracted from diseased plants by means of a handpress and placed in 50-cc. flasks that were held at 20° to 24° C. At intervals, 2-cc. quantities of juice were removed and used to inoculate plants of *Chenopodium capitatum*. Results were based on local-lesion counts of inoculated leaves. The results of three tests, selected as representative, are shown in table 8. Tests were made also with 13

TABLE 8.—*Properties of the sugar beet yellows virus as indicated by results of local lesion counts (average of 3 leaves) after mechanical inoculation of Chenopodium capitatum*

Thermal inactivation				Longevity in vitro				Tolerance of dilution			
Temperature, (° C. for 10 min.)	Lesions per leaf in trial No.—			Period aged at 20° to 24° C.	Lesions per leaf in trial No.—			Dilution	Lesions per leaf in trial No.—		
	1	2	3		1	2	3		1[1]	2[2]	3[3]
	Number	*Number*	*Number*	*Hours*	*Number*	*Number*	*Number*	*Parts*	*Number*	*Number*	*Number*
Check, 20–24	83. 3	14. 0	9. 0	0	34. 6	133. 2	66. 6	0	6. 0	53. 0	337. 5
45	16. 9	6. 3	2. 1	4	3. 3	88. 0	18. 0	1 to 10	7. 0	15. 5	148. 5
50	. 7	. 7	. 3	8	1. 0	29. 3	20. 0	1 to 100	5. 5	9. 5	36. 0
55	0	0	0	12	. 3	1. 5	8. 0	1 to 500	1. 0	8. 5	45. 0
60	0	0	0	24	0	0	2. 0	1 to 1,000	0	3. 5	9. 5
				48	0	0	0	1 to 5,000	0	1. 0	7. 0
								1 to 10,000	0	0	0

[1] Juice from sugar beet.
[2] Juice from *Chenopodium murale.*
[3] Juice from *Nicotiana clevelandii.*

other lots of juice from 4 species of plants. In most tests, the virus was inactivated in 12 hours or less. In one test, there was some survival after 24 hours.

Tests with juice from sugar beet, New Zealand spinach, *Nicotiana clevelandii*, and *C. capitatum* indicated that the source of inoculum did not appreciably influence the period of survival of the virus in extracted juice, despite the fact that juice from *N. clevelandii* appeared to have a much higher titer at the beginning of the test. It appears from the results of all tests that the virus content of expressed juice decreases very rapidly and may be lost in 8 hours and rarely survives more than 24 hours. These periods of survival are considerably shorter than those found by Bercks and Zimmer (*9*) when they used serological methods. It may be, however, that the virus retains its serological properties for longer periods than it retains its capacity to produce infection.

Other Properties

Juice from diseased plants was dried in watchglasses and resuspended in water equal to the original volume of juice after different time intervals and used to inoculate leaves of *Chenopodium capitatum*. These tests indicated that the virus did not survive drying in plant juice. Also, no virus was recovered from dried leaves of diseased plants. However, the virus was active in frozen juice of *Chenopodium murale* and *C. capitatum* after 12 months. The amount of infection obtained indicated no appreciable reduction in virus concentration of the juice during this period.

STRAINS OF THE YELLOWS VIRUS

The virus causing yellows of sugar beet in the United States is a complex of strains differing in virulence and other characteristics. The dominant types of yellowing in all areas where the disease has been studied are typical of the yellowing produced by strains of low to intermediate virulence. In some areas a part of the milder form of yellowing in beets is caused by the radish yellows virus described by Duffus (*21*). The relative importance of mild strains of yellows virus and radish yellows virus in the production of the less severe forms of yellowing of beets has not been determined.

The most virulent strains of beet yellows virus recovered have come from the Salinas, San Joaquin, and Imperial Valleys of California, and the Salt River Valley of Arizona. Plants infected with virulent strains appear to be increasing in incidence in all these areas.

Attempts were made to isolate and purify variants that more or less represent the range of virulence found within the beet yellows virus complex. In this work, sugar beet, *Chenopodium murale*, *C. amaranticolor*, *C. capitatum*, *Nicotiana clevelandii*, and New Zealand spinach have been used extensively as differential hosts. Of these plants, sugar beet, New Zealand spinach, and *C. capitatum* have given the widest range of symptoms. For the sake of simplicity in nomenclature the isolates that have been selected and preserved are designated as "strains" in this publication.

Description of Selected Strains

Probably variants of the yellows virus are so numerous that efforts to isolate and describe all of them would be impractical. Therefore, attempts to separate and describe strains of the yellows virus complex have been limited to isolates that produce characteristics on differential hosts that permit identification with a reasonable degree of certainty.

Six of the selections that have been made have been under observation for more than 3 years. These strains have been numbered 1 to 6, inclusive, as they were selected. Three of the strains do not produce vein clearing on sugar beet and range from very low to intermediate in virulence. Three produce vein clearing on sugar beet and are considered more virulent. The six strains have remained reasonably stable in both sugar beet and New Zealand spinach over a period of more than 2 years, but it has not been established with certainty that the selections represent single entities. Attempts have been made to purify further the three vein-clearing strains by successive transfers on sugar beet by means of single aphids used in short-period feedings. In the course of these purification attempts, avirulent isolates were obtained from two of the vein-clearing selections. The three less virulent strains have not changed perceptibly in any of the hosts in which they have been held.

The six virus isolates that have tentatively been called strains may be divided into two groups and characterized as follows:

Group 1.—These strains do not produce vein clearing on sugar beet, New Zealand spinach, or *Nicotiana clevelandii;* symptoms consist mainly of yellowing of older leaves of infected plants. They are not lethal on plants of *Chenopodium capitatum*, and they have not been transmitted by juice inoculation. (Strains 1, 3, and 6.)

Strain 1. Distinguished from strain 3 by its ability to produce mild mottling on young leaves of plants of New Zealand spinach.

Strain 3. More virulent than strain 6; does not produce mottling on young leaves of New Zealand spinach.

Strain 6. Very mild on all hosts. Produces no symptoms on New Zealand spinach and only slight reddening of older leaves of *C. capitatum* after 20 to 30 days. No symptoms on *C. amaranticolor*, *C. murale*, or *Beta macrocarpa*.

Group 2.—These strains produce distinct vein clearing on sugar beet, New Zealand spinach, and *Nicotiana clevelandii*, and mottling on *Chenopodium amaranticolor*. Usually these strains are lethal on *C. capitatum*. Small necrotic lesions are produced on leaves of *C. capitatum* after inoculation with juice of diseased plants. (Strains 2, 4, and 5.)

Strain 2. Somewhat milder than strains 4 and 5 on all tested hosts.

Strain 4. Greater tendency to produce pinpoint lesions on beet leaves than strains 2 and 5.

Strain 5. Somewhat more severe on beet, and possibly other hosts, than strain 2. Probably more readily transmissible by means of individual green peach aphids and by mechanical inoculations than strains 2 and 4.

Cross-Protection Tests

In general, plants infected and completely invaded by one strain of a virus are immune or highly resistant to infection with a second strain of the same virus. However, Giddings (*24*) showed that strains of the curly top virus do not offer protection against either infection or injury by other strains of the virus, and Bennett (*5*) showed that this is true also with strains of curly top virus in *Samolus parviflorus* (water pimpernel).

Cross-protection tests were made with two virulent strains (strains 2 and 5) and two less virulent strains (strains 3 and 6) of the yellows virus to determine whether evidence of strain interference or cross-protection could be detected. Both virulent strains produce marked vein clearing on young beet leaves, usually in 7 to 12 days after inoculation, whereas the less virulent strains produce symptoms later that consist mainly of mild yellowing of foliage, usually confined to the older leaves. Very little yellowing is produced by strain 6 under greenhouse conditions.

Strains 2 and 3 were used in the first test. Beet plants were inoculated with each strain in the cotyledon stage. Forty-two days later, after the plants had grown to considerable size, plants that had shown definite symptoms characteristic of the respective virus strains were selected and reinoculated with the two virus strains in the four possible combinations. At the same time healthy plants of the same age were inoculated with each of the two strains. In the second test, strains 5 and 6 were used in a similar way and the second inoculation was made 49 days after the first.

A summary of the results of these two tests is shown in table 9. All plants inoculated for the first time became infected and showed the type of symptoms characteristic of the strain used. All plants infected with either of the less virulent strains and later inoculated with either of the virulent strains began to develop distinct vein clearing in the young leaves a few days after the second inoculation. This was followed by production of yellow, thickened, and brittle older leaves. There was no evidence that reinoculation of any of the infected plants with either of the less virulent strains resulted in the production of more severe symptoms.

Further tests were made in which plants of New Zealand spinach were inoculated with strains of low virulence and reinoculated 1 to 2 months later with strains of high virulence that produce vein clearing. As the strains of low virulence used do not produce vein clearing on New Zealand spinach and the more virulent strains produce vein clearing on the leaves as long as the plant continues to grow rapidly, the presence of a superimposed vein-clearing strain is easily detected in this species. In these tests, there appeared to be no protection offered by strains of low virulence against infection and injury by vein-clearing strains.

Under the conditions of these tests little or no protection was afforded by strains of low virulence against infection and injury by more virulent strains of the virus in sugar beet and New Zealand spinach. More evidence is required, however, before final conclusions may be reached regarding the degree to which yellows virus strains protect, or fail to protect, each other. Watson (*56*) reported

TABLE 9.—*Results of cross-protection tests with virulent strains 2 and 5 and less virulent strains 3 and 6 of yellows virus*

[First inoculation made in the cotyledon stage and the second 42 or 49 days later]

Strain of virus used in first inoculation	Strain of virus used in second inoculation	Plants inoculated	Final average severity of symptoms [1]
		Number	*Grade*
3	3	5	1. 4
3	--	5	1. 8
3	5	10	4. 3
--	3	5	1. 2
5	--	5	4. 0
5	3	5	3. 2
--	5	5	4. 6
--	--	[2] 5	0
6	6	5	1. 0
6	--	5	1. 0
6	2	10	3. 3
--	6	5	1. 0
2	--	5	3. 2
2	6	5	3. 2
--	2	5	3. 2
--	--	[2] 5	0

[1] Based on numerical system of grading, which ranged from 1 to 9, inclusive, in ascending order of severity of yellowing.
[2] Check.

a degree of cross-protection between yellows virus strains in England in tests made by juice inoculation. Also, Canova (*13*) reported that attempts to superimpose a "Romagna" yellows virus on sugar beets already infected with the common form in Italy were unsuccessful. It seems probable, therefore, that a certain degree of antagonism may exist between certain strains of yellows virus, although no evidence has been obtained indicating that this is true with isolates that are considered strains of the yellows virus in the United States.

The uncertainty with respect to the cross-protection relationships of viruses and virus strains that have been studied in different parts of the world on sugar beet is further emphasized by the discovery by Russell (*46*) in England and Duffus (*21*) in the United States of viruses, apparently distinct from true yellows virus, that cause yellowing of beets not easily distinguishable from yellowing caused by the less virulent strains of beet yellows virus.

RELATION OF YELLOWS VIRUS TO VECTORS

The relationships of the yellows virus to the green peach aphid has been studied in England by Watson (*54*, *55*). Sylvester (*52*) reported results of similar studies in the United States. These studies have shown that the virus is a "persistent" or "semipersistent" type in relation to the green peach aphid and that acquisition and transmission of yellows virus by this insect is more or less in accordance with expectations from this type of relationship.

In the course of work on the yellows disease reported in this bulletin, further studies were made of some of the relationships of the yellows virus to certain of its vectors. Virus strain 5 has been used in all tests. This strain has the advantage of producing distinct vein clearing in young leaves of beet plants in 7 to 12 days which facilitates the reading of results, and probably increases the accuracy of the findings. Most tests were made with the green peach aphid, but other vectors were used to a limited extent.

Relation of Age and Form of Aphid to Transmission

Alate (winged) green peach aphids and apterous (wingless) individuals of various sizes were tested to determine their relative effectiveness in transmission of the yellows virus. The aphids were colonized on rapidly growing sugar beet plants infected with virus strain 5. After the aphids were well established, leaves were selected that carried large numbers of aphids in different stages of development. The leaves were allowed to wilt until the aphids detached their mouth parts and began to move. They were then lifted with a small brush and placed on seedling sugar beet plants, one aphid being placed on each plant. The four types of aphids tested were classified as (1) small (very young), (2) medium size, (3) adult wingless, and (4) adult winged. Except in some cases with the winged form, all aphids in a single test were taken from the same beet leaf. In each test, 20 plants were inoculated with each type of aphid. In some tests the aphids were placed directly on the plants and each lot of plants was kept in a separate cage 24 hours, after which the insects were destroyed. In other tests aphids were confined on leaves in leaf cages for 24 hours and then removed.

The test was replicated 12 times. The results were paired in the order in which the tests were made. The sums of the paired tests are presented in table 10 as percentages of infection. There was considerable variation in results of different tests, but analysis by the arc-sine transformation method of analysis of variants showed that some of the results are highly significant. Medium-size and adult

TABLE 10.—*Yellows infection produced on seedling sugar beets by green peach aphids of different ages or form*

Test No.	Plants infected of 40 plants inoculated by aphids of indicated size or form (1 aphid per plant)			
	Small aphids	Medium-size aphids	Adult wingless aphids	Adult winged aphids
	Percent	*Percent*	*Percent*	*Percent*
1	15.0	17.5	30.0	15.0
2	5.0	7.5	32.5	10.0
3	7.5	15.0	20.0	2.5
4	20.0	25.0	27.5	5.0
5	22.5	22.5	17.5	10.0
6	15.0	27.5	20.0	12.5
Average	14.2	19.2	24.1	9.2

wingless aphids showed superiority over winged aphids at the 1-percent level, and wingless adults were superior to small aphids at the 5-percent level. Whether these differences reflect differences in innate ability to transmit the virus remains to be determined. It was observed that the winged aphids were more restless than the other types and appeared to feed less. It is possible that this may be one of the factors involved in the lower efficiency of the winged aphids in transmission of virus in these tests.

Feeding Time Required for Aphids to Acquire Virus

Tests were made to determine the relation between the time the green peach aphid feeds on yellows plants and its ability to transmit the yellows virus to healthy plants. In these tests nonviruliferous aphids were placed on leaves of diseased beet plants and allowed to feed for different periods, which ranged from 5 minutes to 72 hours. At the termination of the feeding period the aphids were transferred immediately to healthy seedling sugar beet plants and allowed to feed 24 hours. They were then destroyed and the plants were watched for the appearance of symptoms of yellows. Three adult, or nearly adult, wingless aphids were placed on each plant, and 20 plants were inoculated in each test at each time interval.

The results of five replications of this test are shown in table 11. There was no evidence that the aphids were able to pick up the yellows virus in a feeding time of 5 minutes. Apparently very little virus was acquired in feeding periods of 10 and 20 minutes. It was not until the aphids had fed 1 hour or more that they acquired sufficient virus to produce an appreciable amount of infection. A 6-hour feeding period seemed to enable the aphids to reach their maximum effectiveness as vectors, and feeding periods longer than this apparently did not increase further their virus charge.

TABLE 11.—*Relation of feeding period of green peach aphids on diseased plants to subsequent infection of healthy seedling sugar beet plants*

Experiment No.	Plants infected of 20 inoculated by aphids that had been feeding on diseased sugar beet plants for—										
	5 minutes	10 minutes	20 minutes	1 hour	2 hours	4 hours	6 hours	12 hours	24 hours	48 hours	72 hours
1	0	1	0	3	2	----	15	12	15	12	8
2	0	0	0	0	8	----	8	2	5	2	0
3	0	0	1	10	13	----	19	14	17	19	14
4	0	0	0	1	2	5	12	14	14	15	15
5	0	0	0	0	10	----	4	6	14	12	13

Feeding Time Required for Aphids to Transmit Virus

Tests to determine the relationship of period of feeding of viruliferous aphids on seedling sugar beet plants to amount of infection pro-

duced were made with green peach aphids that were fully charged with virus strain 5. The aphids were placed on healthy seedling beets and allowed to feed the allotted time. They were then destroyed and the plants were watched for the appearance of symptoms of virus yellows. Three aphids were used to inoculate each healthy plant, and 20 plants were inoculated at each time-interval in each test.

The results of three replications of this test are shown in table 12. The aphids produced infection in a feeding time of 5 minutes, but infection was relatively low in the 5-, 10-, and 20-minute feeding periods. In two tests, percentage infection rose to a level in the 1-hour feeding period that was maintained in the remaining feeding periods. In the third test a high percentage of infection was obtained in the 12-hour and longer feeding periods. The reasons for these variations have not been determined.

TABLE 12.—*Relation of feeding period of viruliferous green peach aphids on healthy seedling sugar beet plants to infection*

Experiment No.	Plants infected of 20 inoculated by viruliferous aphids that had been feeding on test plants (3 aphids per plant) for—									
	5 minutes	10 minutes	20 minutes	1 hour	2 hours	6 hours	12 hours	24 hours	48 hours	72 hours
1	1	2	7	15	14	18	17	18	14	12
2	1	3	6	11	10	14	.....	16	16	15
3	0	0	0	8	8	6	14	15	12	12

Time That Virus Is Retained by the Green Peach Aphid

Green peach aphids were removed from sugar beet plants infected with virus strain 5 and placed in cages on radish plants. As radish is immune to infection with the strain of yellows virus used, the time the viruliferous aphids were able to retain the virus while confined to this plant should be a measure of the time the virus is able to persist in the aphid. Leaves with aphids were removed from the radish plants at 24-hour intervals, and 10 or more aphids from the radish leaves were placed on each of 20 seedling sugar beet plants. The aphids were allowed to feed on the beet plants 48 hours, and then destroyed. The plants were watched for appearance of yellows symptoms.

The results of five replications of this test, shown in table 13, are somewhat erratic and suggest that environmental conditions to which the aphids were subjected while feeding on radish, or other factors, may have influenced the period of persistence of the virus in the vector. The results indicate that the virus content of the aphids decreased rather rapidly and that the virus was able to persist in the aphid 72 hours in some instances, but not for 96 hours.

TABLE 13.—*Time the green peach aphids retained the yellows virus while feeding on an immune host plant (radish)*

Test No.	Plants infected by direct transfer of aphids from diseased beets to 20 seedlings	Sugar beet seedlings infected of 20 inoculated by aphids [1] from yellows sugar beet plants after feeding on an immune plant for—			
		24 hours	48 hours	72 hour	96 hours
	Number	*Number*	*Number*	*Number*	*Number*
1	19	7	6	1	0
2	11	2	0	0	0
3	20	9	0	0	0
4	20	0	0	0	0
5	20	13	8	2	0

[1] 10 or more aphids were placed on each seedling beet plant.

Relative Efficiency of Myzus persicae and Aphis fabae as Vectors of the Yellows Virus

The two species of aphids that occur most abundantly on sugar beet are *Myzus persicae* and *Aphis fabae*. Both species are considered important agents in the spread of the yellows disease, but their relative abundance and importance as vectors vary widely in different areas and even in different seasons of the year.

Tests were made to determine the relative efficiency of the two species as vectors under greenhouse conditions. In these tests, the two species were colonized together on the same diseased beet plants. Comparisons were made with adult wingless individuals from plants infected with virus strain 5. Aphids were transferred by means of a small brush from diseased leaves to healthy seedling plants, where they were permitted to feed 24 hours, and then removed. The individuals of the two species came from the same diseased beet leaf in each test. Inoculations were made with 1, 2, 5, and 10 aphids.

The results of these tests, shown in table 14, indicate that *Myzus persicae* is a much more efficient vector of the yellows virus used than *Aphis fabae*. This is especially evident in the tests in which inoculations were made with one aphid per plant. The reduced infection in plants inoculated with *A. fabae* apparently was not associated with less severe symptoms of the disease. Average grade of severity of symptoms was approximately the same in plants infected by means of each of the two species of aphids.

Further tests were made with 1 aphid per plant in which 188 plants were inoculated with each species of aphid. In these tests, *M. persicae* produced infection in 37 plants, whereas *A. fabae* produced infection in only 12 plants.

In other tests the potato aphid, *Macrosiphum solanifolii*, was compared with *M. persicae*. This aphid feeds readily on sugar beet and breeds on the plant in the greenhouse, but it is not often found on sugar beet under field conditions. Results indicate that its efficiency

TABLE 14.—*Comparison of Myzus persicae and Aphis fabae as vectors of the yellows virus*

Test No.	Aphid tested	Plants infected of 20 inoculated with the indicated number of aphids			
		1 aphid	2 aphids	5 aphids	10 aphids
		Number	*Number*	*Number*	*Number*
1	*Myzus persicae*	12	16	19	16
	Aphis fabae	0	5	7	9
2	*Myzus persicae*	11	16	20	20
	Aphis fabae	3	13	11	15
3	*Myzus persicae*	13	18	18	18
	Aphis fabae	4	2	4	8
4	*Myzus persicae*	3	6	12	18
	Aphis fabae	0	2	3	15

as a vector of the yellows virus is considerably below that of *M. persicae* and probably in the range of that determined for *A. fabae.*

Conclusions

Transverse sections of beet leaves on which green peach aphids have fed indicate that the path of penetration of the stylets is not direct, as with the beet leafhopper, but somewhat crooked and inclined to follow to some extent the intercellular spaces. Apparently, penetration is not so rapid as that effected by the beet leafhopper. All evidence indicates that the green peach aphid feeds predominantly in the phloem. The method of feeding probably precludes the acquisition of appreciable quantities of virus from parenchyma tissue, even if the virus were present in high concentrations in such tissues. It seems probable, therefore, that most of the virus acquired by the vector is derived from the phloem. This concept is supported by the rather abrupt increase in the efficiency of the green peach aphid as a vector after feeding periods of 20 minutes to 1 to 2 hours on diseased plants, as shown in table 11.

The retention of the yellows virus by the aphid for periods of 72 hours while feeding on an immune host suggests that the virus is retained largely in the body of the insect, perhaps in the blood, rather than as a contaminant on the mouth parts. The loss of virus in periods longer than 72 hours suggests that the virus does not multiply in the vector. In summary, the evidence suggests that the yellows virus is picked up by the aphid principally from the phloem and that it passes into the alimentary tract with the ingested food: from the alimentary tract it passes into the blood, from which it passes into the salivary glands and is reintroduced into the plant through the medium of the saliva. The period of retention in the insect may represent the time required for exhaustion of the virus charge, but, more likely, it represents the time the virus is able to retain its activity outside of the living plant.

Aphids undoubtedly introduce yellows virus into all types of tissues penetrated by their stylets. There may be considerable differences,

however, in the susceptibility to infection of the different tissues into which the virus is introduced. The results shown in table 12 indicate that the green peach aphid is able to produce infection in a feeding time of 5 minutes. This would indicate that systemic infection can be produced by introduction of the virus into parenchyma, as the aphid probably could not often penetrate to the phloem in so short a time. The rather marked increase, however, in the percentage of infection in the 1-hour feeding period as compared to shorter periods suggests that infection may be much more certain if the virus is introduced into the phloem by the aphid.

RELATION OF THE YELLOWS VIRUS TO THE PLANT

Plant viruses probably invade all types of living tissue, but the extent of invasion varies with the virus and plant involved. In some instances there appears to be a high degree of tissue specificity. Those viruses that produce disturbances primarily in the parenchyma probably occur in greatest concentration in that tissue. Usually such viruses produce mottling or other types of chlorotic or necrotic spotting. Some viruses that produce disturbances primarily in the vascular system appear to be more highly concentrated in the phloem. In general, such viruses induce leaf curling, yellowing, or rosetting, but they rarely produce mottling.

Sugar beet yellows has certain characteristics of both the "mosaic" and the "yellows" groups of diseases. Chlorotic and necrotic spots are produced in beet leaves by some strains of the virus, and definite mottling is evident in leaves of *Chenopodium amaranticolor*. The general appearance of the disease in the field, however, is more typical of the yellows group of diseases. Studies have been made to determine some of the relationships of the yellows virus to its host plants as well as some of its relationships to specific tissues.

Relation of Point of Inoculation and Age of Plant to Infection

Tests have shown that beet leaves of all ages are susceptible to infection with the yellows virus, but, as yet, no tests have been made with leaves of different ages on large plants to determine their relative susceptibility to infection. Tests were made, however, to determine relative susceptibility of cotyledons and true leaves of beet plants in the two-leaf stage of development in which viruliferous green peach aphids were caged singly on cotyledons and true leaves. The results of 5 replications of this test, involving 200 plants in each treatment, indicated that there was no significant difference between the susceptibility of cotyledons and true leaves to infection when the plants were in the 2-leaf stage of development.

Apparently there is little increase in resistance to infection with increase in age of the beet plants. Plants in the cotyledon stage compared with plants 5 weeks after potting have given approximately the same percentages of infection when inoculated by means of one aphid per plant. In field inoculations over a period of 5

years, nearly 100 percent infection with more virulent strains has been obtained with approximately 10 aphids per plant, regardless of the size of the plants at the time of inoculation.

In 1956, for instance, plots planted December 15, 1955, were inoculated March 26, April 30, and June 5 with approximately 10 aphids per plant. Each plot had approximately 300 plants, and each date of inoculation was replicated 8 times. Infection was above 95 percent in all plots, and there was no obvious difference in the amount of infection produced on the different dates. The results of this series of inoculations indicate that large beet plants retain a very high degree of susceptibility to infection.

Rates of Movement of Yellows Virus Out of Inoculated Leaves

The time required for the movement of a virus out of an inoculated leaf is influenced to a large degree by the tissue into when the virus is introduced initially. Curly top virus probably is introduced directly into the phloem of beet plants through the feeding of the beet leafhopper (*Circulifer tenellus* (Baker)), and the virus is known to move out of the inoculated leaf very rapidly. Rates of movement as high as 1 inch per minute have been recorded (*2*). Viruses that are introduced into the parenchyma through juice inoculation are known to require longer periods of time in which to escape from the leaf into which they are inoculated.

Tests were made to determine the rates of movement of the yellows virus out of inoculated leaves of sugar beet and of *Chenopodium capitatum* after the introduction of the virus by means of the green peach aphid and by means of mechanical inoculation with plant juice.

Movement Out of Beet Leaves

To determine the rate of movement of yellows virus out of beet leaves following introduction by means of a natural vector, viruliferous green peach aphids were caged on the distal ends of leaves of rapidly growing sugar beet plants. In the tests all inoculated leaves were more than 10 cm. long at the time of inoculation. After each period of aphid feeding the inoculated leaf on half of the plants was severed 10 cm. from the point of inoculation. At the same time the aphids were removed from the inoculated leaf of the remaining plants and the plants were retained to provide a check on the probable amount of infection produced by the aphids feeding on the inoculated leaves that were removed.

The results of this test, shown in table 15, indicate the number of plants infected in the indicated feeding period and the number of infected plants in which the virus moved a distance of 10 cm. from the point of introduction in the allotted time interval.

No virus moved out of the inoculated leaf in a period of 10 minutes, although the results with the check plants indicated that some of the inoculated leaves probably were infected. The virus moved out of 6 of 67 leaves in a period of 20 minutes. In general, the percentage of leaves in which the virus moved a distance of 10 cm. increased with the time interval, but there was some variation, probably caused

TABLE 15.—*Rates of movement of yellows virus out of inoculated leaves of sugar beet plants following introduction of virus by means of green peach aphids*

Time for virus to move 10 cm.	Results in plants from which inoculated leaf was removed		Results in plants from which inoculated leaf was not removed	
	Plants inoculated	Infection	Plants inoculated	Infection
	Number	*Percent*	*Number*	*Percent*
10 minutes	76	0. 0	76	9. 2
20 minutes	67	9. 0	65	30. 8
30 minutes	66	9. 1	64	35. 9
40 minutes	99	11. 1	95	49. 5
1 hour	104	9. 6	97	53. 6
2 hours	94	10. 6	90	74. 2
3 hours	64	10. 9	60	81. 7
4 hours	81	23. 5	78	85. 9
6 hours	45	28. 9	42	76. 2
8 hours	56	17. 9	41	73. 2
12 hours	50	16. 0	50	95. 0
24 hours	71	33. 8	68	79. 4
48 hours	40	42. 5	40	82. 5
72 hours	30	83. 3	30	93. 3

in part by the fact that not all inoculations were made at the same time and under the same conditions.

The results with check plants and plants from which the inoculated leaf was removed indicate rather clearly that the virus probably did not move out of all the leaves that were infected even in periods of 24 and 48 hours.

Efforts were made to determine rates of movement of yellows virus out of inoculated beet leaves after introduction of the virus by mechanical inoculation: however, these tests were abandoned, owing to the difficulty of obtaining infection on sugar beet plants by mechanical inoculation. It was thought, however, that essentially the same results might be obtained by using beet mosaic virus, which is readily juice-transmissible.

Beet plants growing rapidly in boxes were selected for the test. One leaf on each plant was inoculated by rubbing inoculum from mosaic plants over the surface of the leaf tip. At different time intervals following inoculation, the inoculated leaf of approximately three-fourths of the plants was severed at a distance of either 10, 20, or 30 cm. from the inoculated area and the inoculated leaf on the remaining plants was retained. Thus, the experiment was designed to determine the time required for the virus to move a distance of 10 cm. out of the leaf blade, which in most instances was less than 10 cm. long, and also an additional 10 or 20 cm. through the petiole.

The results of this test, shown in table 16, indicate that the beet mosaic virus required more than 48 hours to move a distance of 10 cm. out of the inoculated leaf, and in the majority of plants it required more than 72 hours. The results indicated that whether the virus was

TABLE 16.—*Influence of distance of travel of beet mosaic virus on percentage of systemic infection in beet plants following mechanical inoculation and removal of inoculated leaves 10, 20, and 30 centimeters from points of inoculation at 6-hour intervals*

Time for virus to move out of inoculated leaf (hours)	Distance of movement of infection out of leaf					
	10 cm.		20 cm.		30 cm.	
	Plants inoculated	Infection	Plants inoculated	Infection	Plants inoculated	Infection
	Number	*Percent*	*Number*	*Percent*	*Number*	*Percent*
48	42	0. 0	40	0. 0	42	0. 0
54	40	2. 5	41	0	40	0
60	64	3. 1	67	4. 5	66	3. 0
66	65	4. 6	64	6. 2	64	6. 2
72	53	3. 8	59	6. 8	51	3. 9
78	53	39. 6	55	25. 4	50	34. 0
84	43	55. 8	49	44. 9	43	41. 9
Check [1]	51	84. 3	62	69. 4	61	78. 7

[1] The inoculated leaves were not removed.

required to move 10, 20, or 30 cm. to produce systemic infection made little, if any, difference in the number of plants showing systemic infection. It seems probable, therefore, that there was first a slow movement of virus through the parenchyma before the virus enters the phloem, but that after entrance to the phloem was effected, movement was so rapid that additional movements of 10 to 20 cm. through the petiole could not be detected in the 6-hour intervals of the test.

Movement Out of Leaves of Chenopodium capitatum

The susceptibility of *Chenopodium capitatum* to infection from inoculation with plant juice afforded an opportunity to compare movement of the yellows virus in the same species of plant after the introduction of the virus by aphids and by mechanical inoculation.

In tests with a vector, 10 viruliferous green peach aphids were allowed to feed on the distal end of a leaf of each inoculated plant of *C. capitatum* for time-intervals ranging from 10 minutes to 6 hours. At the end of the allotted feeding period the inoculated leaf on half of the plants was severed 10 cm. below the point of inoculation. At the same time, the aphids were removed from the inoculated leaves of the remaining plants, which served as checks on the amount of infection produced by the feeding of the aphids.

The results of this test, shown in table 17, indicate that the virus moved out of the inoculated leaf of one of five plants in a period of 40 minutes. A period of 6 hours was sufficient to permit movement out of all inoculated leaves. The results in the check plants indicate that infection was produced in a feeding time of 10 minutes and that percentage of infection was high in longer feeding periods.

TABLE 17.— *Rate of movement of yellows virus a distance of 10 cm. out of inoculated leaves of Chenopodium capitatum as influenced by method of inoculation*

Time for virus to move 10 cm.	Method of inoculation	Results in plants from which inoculated leaf was removed		Results in plants from which the inoculated leaf was not removed	
		Plants inoculated	Infection	Plants inoculated	Infection
		Number	*Percent*	*Number*	*Percent*
10 minutes	Aphids	4	0. 0	5	20. 0
20 minutes	do	5	0	5	80. 0
30 minutes	do	5	0	5	60. 0
40 minutes	do	5	20. 0	5	100. 0
1 hour	do	5	0	5	80. 0
2 hours	do	5	20. 0	5	80. 0
3 hours	do	5	40. 0	5	100. 0
4 hours	do	5	60. 0	5	100. 0
6 hours	do	5	100. 0	5	100. 0
2 days	Juice	8	0	8	25. 0
3 days	do	8	0	8	37. 5
4 days	do	8	12. 5	8	37. 5
5 days	do	8	0	8	25. 0
6 days	do	8	12. 5	8	37. 5
7 days	do	8	25. 0	8	25. 0
10 days	do	8	12. 5	8	37. 5
14 days	do	8	25. 0	8	50. 0

This experiment was repeated, except that the inoculations were made by mechanical inoculation and three leaves were inoculated on each plant. Numerous local lesions were obtained on all leaves that were retained for a sufficient length of time, and systemic infection was reasonably satisfactory in the check plants, as shown in table 17. There was no movement of virus out of inoculated leaves until the fourth day. More extensive tests probably would indicate a shorter period for movement out of the inoculated leaves, but the fact that there was no movement out of inoculated leaves in the 1-, 2-, and 3-day periods indicates that the virus required considerable time to move through the parenchyma from points of introduction and to enter the phloem.

The results from juice inoculation of *Chenopodium capitatum* indicate that when the yellows virus is introduced into the parenchyma of the leaf of a susceptible plant it moves very slowly through the intervening parenchyma cells and eventually enters the phloem through which it presumably moves very rapidly. The rate of movement through the parenchyma after juice inoculation is more or less the same as that found with beet mosaic virus. The results of inoculation of both beet and *C. capitatum* by means of the green peach aphid indicate rather clearly that the aphid is capable of introducing the yellows virus directly into the phloem. However, the reduced infection in the shorter time-intervals in plants with inoculated leaves removed as

compared with the check plants suggests that some of the virus introduced by the aphids, and that eventually led to systemic infection, was introduced into the parenchyma. It seems probable, therefore, that the aphids introduce yellows virus into all the tissues in which they feed and that infection may result from introduction of virus into either parenchymatous or phloem tissue. Apparently, however, local lesions do not result from introduction of virus into parenchyma by the aphid, whereas local lesions often are quite evident after the introduction of the virus into parenchyma by the rubbing method of inoculation.

Concentration of Virus in the Beet Plant

Tests were made with aphids to obtain information on the relative concentration of virus in leaves of different ages in chronically infected sugar beet plants. Aphids were removed from (1) old leaves, (2) recently matured leaves, (3) half-grown leaves, and (4) small leaves, and caged singly on seedling plants. Nine tests, each involving 20 plants inoculated from each virus source, were made. Infection obtained was 23.5, 34.0, 30.0, and 29.0 percent, respectively, from the different-aged leaves. These results indicated that mature leaves were significantly higher in virus content than old leaves, but other results were not significant.

More extensive tests, using a local-lesion technique, were made to obtain additional information on virus concentration of leaves of different ages at different times after inoculation. In these tests, juice was pressed from the material to be tested and rubbed over leaves of *Chenopodium capitatum* on which a small amount of carborundum had been sprinkled. Tests were made of old, recently mature, and young leaves of chronically infected plants. In further tests comparisons were made between leaves showing vein clearing and leaves of the same age on chronically infected plants showing no vein clearing, and also between the yellow and green areas of leaves from recently infected plants showing sectoring.

The results of these tests are shown in table 18. They indicate that in chronically diseased plants virus concentration is appreciably higher in recently mature leaves than in young leaves of the same plant. The largest contrast, however, was between virus content of young leaves in recently infected and chronically diseased plants. Leaves showing vein clearing had a very high virus titer, whereas leaves of the same age from plants chronically diseased had a relatively low virus titer.

Virus concentration was high also in the yellow areas of leaves showing sectoring. No virus was recovered from the green areas of 8 of 17 leaves; this finding would indicate perhaps that these areas were uninvaded at the time of test. Virus content of the green segments of the remaining nine leaves was low as compared with the yellow segments of the same leaves. Virus concentration in the yellow portions compares favorably with that in young leaves with vein clearing, and it seems probable that high initial concentrations of virus are present in obviously affected areas of segmented leaves.

TABLE 18.—*Relative virus concentration in leaves of different ages on chronically diseased plants, young leaves of recently infected plants showing vein clearing, and in green and yellow areas of sectored leaves in recently infected plants*

Test No.	Lesions produced on *Chenopodium capitatum* by juice from indicated sources (total on 3 leaves, average of 2 plants)					
	Leaves past maturity, from chronically diseased plants	Leaves mature, from chronically diseased plants	Leaves small, from chronically diseased plants	Leaves small, from recently infected plants (vein clearing)	Green parts of sectored leaves	Yellow parts of sectored leaves
	Number	*Number*	*Number*	*Number*	*Number*	*Number*
1	34. 0	29. 5	13. 0	149 0	1. 0	115. 0
2	6. 0	12. 0	2. 5	73. 0	0	9. 5
3	7. 0	9. 5	13. 0	66. 0	0	44. 0
4	18. 0	33. 5	8. 5	167. 5	2. 5	25. 0
5	27. 0	73. 0	6. 0	90. 5	0	8. 5
6	7. 0	20. 0	4. 5	86. 0	0	22. 0
7	26. 0	31. 5	11. 0	71. 5	0	92. 0
8	33. 0	28. 5	12. 5	154. 0	0	15. 0
9	12. 0	34. 5	13. 5	99. 0	1. 5	129. 0
10	23. 0	20. 0	5. 0	91. 5	1. 5	4. 5
11	27. 0	17. 0	6. 0	138. 0	0	44. 0
12	25. 5	30. 5	5. 5	107. 0	0	20. 0
13	21. 0	50. 0	8. 0	205. 0	1. 0	72. 0
14	76. 5	33. 5	39. 0	100. 5	1. 0	39. 5
15	57. 0	62. 5	33. 0	172. 0	14. 5	114. 0
16	58. 5	61. 5	15. 0	162. 5	5. 5	68. 5
17	82. 5	56. 5	36. 5	154. 5	6. 0	83. 0
18	65. 5	59. 0	54. 0	137. 5		
19	46. 5	93. 5	55. 5	170. 0		
20	39. 5	54. 5	37. 0	106. 5		
Average	34. 6	40. 5	19. 0	125. 1	2. 0	53. 3

CARBOHYDRATE TRANSLOCATION AND VIRUS MOVEMENT

Evidence has been presented that the curly top virus is closely associated with the phloem and that its rapid rates of movement in the beet plant can be influenced markedly by manipulation of the direction and rate of transportation of carbohydrates (*3*).

The rates of downward movement of yellows virus in beet leaves, already mentioned, indicate that this virus also moves readily and rapidly in the phloem and suggest that virus movement may be influenced by food transport.

In tests to determine whether invasion of the beet plant by yellows virus may be influenced or controlled by manipulation of carbohydrates transported, advantage was taken of the fact that beets can be induced readily to form separate crowns on the same root system.

Beet roots were split into three parts at the crown and the split ex-

tended about 15 cm. into the root. The plants were potted with the crowns widely separated. The crowns on each plant were pruned to produce shoots of approximately equal size. After the top growth was well advanced, one of the three shoots of each plant was inoculated with yellows virus by means of *Myzus persicae*, one was defoliated at the time of inoculation of the first shoot, and one was retained as a check on virus movement.

Similar tests were made by the viruses of sugar beet mosaic and sugar beet curly top, in order to compare the movement of these two viruses with that of the yellows virus.

The results of the tests with the three viruses are shown in table 19. Each virus moved from the inoculated shoot and produced symptoms in the defoliated shoot in about the same time as in the inoculated shoot. In some instances symptoms appeared earlier on the defoliated shoot than on the inoculated shoot. This probably was due to a more rapid growth of leaves on the defoliated shoot. Appearance of symptoms was much delayed in the noninoculated, nondefoliated shoots. Of the three viruses, mosaic virus moved across into the nondefoliated shoot in the shortest time and produced symptoms in an average period of 33.7 days. Yellows virus required an average period of 57.3 days, whereas curly top virus had not produced symptoms in any of the check shoots in a period of 140 days.

TABLE 19.—*Effect of defoliation in triple-crown beet plants on the movement of viruses causing yellows, mosaic, and curly top*

Virus used	Plants tested	Average time for appearance of symptoms on indicated shoot		
		Inoculated, non-defoliated	Noninoculated, defoliated	Noninoculated, non-defoliated
	Number	*Days*	*Days*	*Days*
Yellows	15	12. 5	14. 1	57. 3
Mosaic	18	9. 4	10. 2	33. 7
Curly top	10	16. 3	18. 1	[1] 140. 0

[1] No symptoms of curly top were evident after 140 days, when the test was discontinued.

Essentially the same results were obtained with triple-crown plants following simultaneous introduction of the three viruses into one shoot and defoliation of a second shoot in each plant at the time of inoculation. Each of the three viruses again produced symptoms in the defoliated shoot at about the same time as in the inoculated shoot. Mosaic symptoms appeared in the check—noninoculated, nondefoliated shoot—in an average time of 37 days, and yellows symptoms occurred in an average time of 57 days. Curly top symptoms appeared on 3 of 10 plants in an average time of 110 days, and no symptoms were evident on comparable shoots of the remaining 7 plants after 180 days, when the experiment was discontinued.

It may be suggested that this type of experiment does not necessarily prove that the virus moved from an inoculated into a noninocu-

lated shoot as a result of defoliation, since the virus may have been present in the nondefoliated check shoots but failed to produce symptoms. This seems highly unlikely, as shoots of approximately the same size and age on the same plants did show symptoms following introduction of virus by direct inoculation. However, the following experiment was made to test the validity of this objection. Forty plants, each with two crowns, were selected. One shoot on each of the 40 plants was inoculated with yellows virus. The noninoculated shoots of 20 plants were defoliated, and the noninoculated shoots on the remaining 20 plants were retained as nondefoliated checks. Symptoms of yellows appeared on the inoculated shoots of the 40 plants in an average time of 9.2 days, the longest incubation period being 16 days. Symptoms appeared on the defoliated shoots in an average time of 9.2 days; the longest incubation period was 17 days. No symptoms were evident on any of the noninoculated, nondefoliated shoots after 20 days.

After 20 days the 20 check plants were divided into 2 lots. The noninoculated shoots of 10 plants were defoliated and the root left attached to the noninoculated half. The roots of the remaining 10 noninoculated shoots were severed about 10 cm. below the crown and each noninoculated part was removed to a separate pot. Yellows symptoms appeared on the attached, defoliated shoots in an average time of 3.8 days after defoliation. The detached, defoliated parts were retained for more than 2 months and developed into thrifty plants. Four showed yellows symptoms and six remained free of yellows symptoms.

This experiment was repeated with the curly top virus. Symptoms of curly top appeared on the inoculated shoots of 20 plants in an average time of 12.4 days, with a maximum incubation period of 19 days. They appeared on the defoliated shoots of the same plants in an average time of 13.3 days, with a maximum incubation period of 19 days. Symptoms appeared on check shoots attached to the plant and defoliated 30 days after the plant was inoculated, in an average period of 4.2 days after defoliation. Symptoms appeared on 2 of 10 shoots that were removed, defoliated, and repotted, and the remaining 8 shoots remained free of symptoms.

This evidence appears to show conclusively that yellows virus had not invaded the majority of noninoculated, nondefoliated crowns of dual-crown beets in a period of 20 days and that the curly top virus had invaded not more than 2 of 10 of the noninoculated, nondefoliated parts in a period of 30 days. The rapid development of symptoms in the shoots that were defoliated 20 to 30 days after inoculation of an attached shoot and left attached to the plants probably is caused by the movement of greater quantities of virus from the inoculated part into the shoots than would be introduced by insect vectors.

These results indicate strongly that the movement of all three viruses into noninoculated shoots of the beet plant can be accelerated by defoliation of these shoots and that this acceleration is associated with transportation of carbohydrates. The more rapid movement of mosaic and yellows viruses into nondefoliated, noninoculated shoots may be caused by the ability of these viruses to increase and move slowly through the parenchyma outside the phloem, whereas the curly top virus may be largely restricted to the phloem and dependent on this type of tissue in its movement through the plant.

CONTROL MEASURES

Under favorable conditions the yellows virus spreads rapidly. Extensive spread may take place from limited sources of infection that, in some instances, may be located considerable distances from beetfields. This rapid spread of the yellows disease is caused, in large part, by the efficiency of insect vectors, chiefly the green peach aphid, in the transmission of the virus. In the principal sugar-beet-producing areas of California, aphids live and increase through the winter months and conditions favor large increases in aphid populations in the late winter and spring.

The presence of aphids throughout the year in many beet-producing areas, particularly in California, and the occurrence of sources of infection such as wild and escaped beets, sprouts from roots remaining in harvested fields, and overlapping of crops of successive years, present unusual problems with respect to control of yellows by conventional methods. The size of aphid populations, diversity and variation in planting dates, and type of source of infection in different areas further complicate the problem of control.

In general, possibilities for control of yellows are limited to one or more of four methods. These consist of (1) destruction of aphid vectors by application of insecticides, (2) destruction of sources of infection for initial spread of the virus to beetfields, (3) planting on dates that enable the beets to escape infection, and (4) development and use of varieties resistant to the disease.

Use of Insecticides

Under certain conditions in Europe, spraying beetfields early in the season to kill aphids has been effective in giving a high degree of control of yellows. Best results were obtained by timing one or more sprays to destroy aphids in the spring before they had an opportunity to produce secondary spread from plants infected initially by winged forms that carry the virus into the field from outside sources. Where spring populations of aphids were high, however, control has been reported as unsatisfactory. In large-scale experiments in the Province of Limberg in 1955, Rietberg and Hijner (*43*) reported that two applications of Systox reduced incidence of infection about 50 percent. They concluded that a single application in this area and in eastern Brabant would be profitable. Similar beneficial results from aphicides have been reported in England and in Germany.

In most areas in California where sugar beets are grown extensively, populations of green peach aphids are usually very high during March, April, and May. During this period, large numbers of winged individuals may enter beetfields from outside sources. It is probable that where virus sources are available, the winged form alone is capable of producing high percentages of infection before June 1 in such areas as the Salinas Valley. This conclusion is based on the numbers of winged aphids found in beetfields and on the results of tests in 1955, 1956, and 1957 in which selected plots were sprayed with insecticides at 7- to 10-day intervals from thinning to 4 to 6 weeks before harvest. In all these tests, infection in the sprayed plots increased until it reached nearly 100 percent before harvest.

Location of diseased plants in the plots indicated a random distribution, such as would be expected from aphids moving in from outside sources. In all the spray tests, damage by the disease was reduced, but costs of spray applications were far in excess of the increased value of the crop.

In areas where conditions are favorable for the production of large populations of aphids throughout most of the year, as in the Salinas Valley, it seems unlikely that attempts to control yellows by application of insecticides will be profitable. In other areas, control by use of insecticides may hold greater promise.

In the San Joaquin Valley and most of the other inland areas of California where summer temperatures are high and humidity low, aphid populations may be high in March and April and early May, after which they may drop to very low levels. It is probable that in most seasons little spread occurs in these areas after the middle of May or the first of June. Under these conditions one or two spray applications, properly timed in March or April to destroy aphids after the winged forms have invaded the fields and before they have had time to increase and produce appreciable secondary spread, might greatly reduce infection. However, further experimental results are needed before definite recommendations are given.

Elimination of Sources of Infection

Yellows is likely to be a serious disease of sugar beet in Western United States only in areas where sugar beet or other host plants are present to serve as sources of infection throughout the year. The principal source of infection of new beet plantings appears to be the beet itself. Wild beets or beets escaped from cultivation and growing along fence rows, ditchbanks, and in waste places may be found in most of the beet-producing areas of California. Also, in some of these areas beetfields are not harvested until after the next crop is already started and in some fields the beets are carried through the winter and harvested the following spring. Such fields serve as sources of infection for fields in adjacent areas and probably for fields over an extensive area in some cases.

Fields may serve as sources of infection even after harvest. Many of the smaller beets are left in the field. Usually the beet crop is followed by barley, alfalfa, cotton, or corn. Many of the beets remaining in the field are destroyed in preparation for these crops, but others are buried just deep enough to produce tops that harbor aphids that may move to new plantings. In barley and alfalfa fields in particular, beets may persist well into the following spring and continue to serve as important virus reservoirs over an extended period.

Spinach is susceptible to yellows and is a favorable host plant for green peach aphids and some of the other vectors of the yellows virus. It is grown as a winter crop in the Salinas Valley of California and in certain other areas of Western United States. Spinach often is infected in the fall from beets or other sources and carries the virus through the winter to serve as a source of infection for beets in late winter and early spring.

Weed hosts appear to be of lesser importance as sources of infection, but their full significance remains to be determined. Most weed

hosts are annuals and apparently are not extensively infected under field conditions. These annual hosts apparently are of little importance.

Australian saltbush (*Atriplex semibaccata*), one of the few hosts that are perennial, may be a source of infection. Yellows virus has been recovered from plants growing adjacent to beetfields in the Salt River Valley of Arizona, and evidence indicates that most of the older plants in the Salinas Valley are infected. Australian saltbush undoubtedly harbors virus in a number of areas where yellows occurs on sugar beet. It is a poor food plant for the green peach aphid. The aphid (*Hyalopterus atriplicis*) commonly found in abundance on the species apparently is unable to transmit the yellows virus. It seems unlikely that extensive spread of yellows from Australian saltbush to beets occurs, but the species may be important in carrying virus through periods between crops in areas such as the Salt River Valley of Arizona, where a small amount of infection from saltbush in the fall might be spread extensively by high populations of aphids during the following winter and spring.

At present, it appears that the practice of harvesting one crop before the next crop is planted will delay and reduce infection. Destruction of wild and escaped beets and beets left in the field after harvest should still further delay spread. However, in California and certain other area in Western United States, even after employment of the most effective sanitary measures likely to be achieved on a large scale, enough virus sources remain to provide virus for extensive spread before the first of June in seasons when aphid populations are high. Although sanitary measures may be expected to delay infection and reduce injury, completely satisfactory control is not likely to be achieved in many areas by these measures under the systems of beet production now being employed.

Relation of Date of Planting to Control

Under some conditions in some areas, the date of planting may greatly influence the amount of yellows infection and the period in the development of the plant in which infection occurs. In some valleys in central and southern California aphid populations drop to very low levels with the advent of high temperatures and low humidities in May and June. Plantings made in late April near Riverside, Calif., remained relatively free of infection throughout the season, even when growing adjacent to infected areas; whereas fields planted in December showed a high percentage of infection. Late planting to escape infection, however, probably would not be a profitable method of control, even in areas where it would be effective, owing to lower yields from late-planted beets.

In some areas in California, early planting may result in lower losses from yellows. Plantings in the Salinas Valley usually are made in December and January, although some plantings are made later. The greater amount of yellows infection occurs in April. Evidence indicates that if plants escape infection in the earlier stages of beet development, reduction in yield due to yellows is likely to be correspondingly reduced. Under these conditions percentage loss would probably be less in beets planted in December than in beets planted in

February or later. The degree of effectiveness of this practice, however, is complicated by a number of factors that may vary in different areas and in different seasons. One of the most important involves the date of harvest of the preceding crop. If the harvest period of one crop extends very far beyond the planting date of the next crop, infection in the new crop may occur in the early stages of plant development and injury under such conditions might be increased by early planting.

Resistant Varieties

Some commercial varieties of sugar beets in common use show somewhat more yellowing than others, but there is no evidence that differences in resistance justify selection of any of the common varieties in preference to others on the basis of resistance to yellows alone. Observations and tests of more than 500 varieties and selections have indicated, however, that there is a wide range of susceptibility to injury within the species *Beta vulgaris*. Injury in a number of these selections has resulted in more than 60 percent reduction in yield, whereas reductions in yields of common commercial varieties of sugar beet have usually not exceeded 35 percent. Use of highly susceptible selections in breeding programs might result in appreciable increase in susceptibility of the final selection.

On the basis of preliminary observations, certain selections also appear to be more susceptible to infection than other selections. At least, certain selections in check plots that were sprayed at regular intervals remained relatively free of yellows infection over periods in which other selections showed nearly 100 percent infection. Whether these selections escape infection as a result of being less attractive to aphids or are inherently more resistant to infection remains to be determined.

A breeding program has recently been initiated by the U.S. Department of Agriculture at the U.S. Agricultural Research Station at Salinas, Calif., in an effort to obtain sugar beets with increased resistance to yellows. Considerable time may be required, however, to obtain substantial results, due principally to lack of criteria for accurate determination of resistance in individual selections and to the apparent absence of wide ranges of variation in the resistance in the genetic material available for selection.

Substantial progress in breeding for resistance to yellows has been reported from Europe. In the Netherlands breeding programs have been in progress since 1948 and, although immunity has not been achieved, selections have been developed in which yield reductions from the heaviest infections do not exceed 14 to 16 percent (*43*).

Production of more resistant varieties constitutes the chief hope of satisfactory control of yellows and, in certain areas, this is the only method of control that appears likely to be effective under the climactic conditions that prevail and the methods of production of sugar beets that appear to be most profitable from the standpoint of processing costs and most economical use of land.

SUMMARY

The disease known as beet virus yellows or beet yellows has been present in beetfields in Europe for many years. It was not identified

in the United States until 1951, but it probably was present several years earlier. The distribution of the disease is now practically worldwide in areas that produce sugar beets.

The causal virus has an extensive potential host range. Crop plants that are affected include sugar beet, table beet, Swiss chard, other close relatives of sugar beet, and spinach. The causal virus has been transmitted to at least 66 species of plants representing 12 families. Chief symptoms on sugar beet consist of yellowing of mature leaves, often followed by necrosis of areas between veins, and thickening of the entire leaf. Certain virus strains also produce vein clearing or vein yellowing in young leaves in the earlier stages of development of the disease. Certain of the weed hosts, as chickweed and Australian saltbush, are symptomless carriers of the virus, at least under most environmental conditions.

In areas where extensive infection occurs in early stages of plant development, yellows causes marked decreases in yield of roots. In replicated plot tests at Riverside and Salinas, Calif., the disease reduced yields as much as 47 percent and sucrose content from 0.1 to 3.1 percentage points. Reduction in yield in areas where the disease is most prevalent probably reaches 25 percent or more. Yellows may increase injury by cercospora leaf spot and by curly top in areas where these diseases are prevalent.

The causal virus is transmitted by a number of species of aphids, the most important being the green peach aphid. Seven of 14 species of aphids tested, however, did not prove to be vectors. The virus was not transmitted by species of *Cuscuta*. Tests showed that the virus was present in dodder growing on diseased plants, but the virus was soon lost when the dodder was transferred to plants immune to yellows. The virus was transmitted to sugar beet, to *Chenopodium murale*, and to *C. capitatum* by juice inoculation. Best results were obtained with *C. capitatum* from inoculations during the winter months. Local necrotic lesions, about 1 mm. in diameter, were produced. Production of local lesions was not always followed by systemic infection.

The virus has a thermal inactivation point between 50° and 55° C. At room temperature it was active in plant juice after 24 hours but not after 48 hours. A small amount of infection was obtained from juice diluted 1 to 5,000, but no infection was obtained from dilutions of 1 to 10,000. The virus was active in frozen plant extracts after 12 months.

The yellows virus exists in the United States as a complex of strains, ranging in virulence from those that produce mild yellowing on older leaves of sugar beet to strains that produce vein clearing on young leaves and marked yellowing and necrosis on older leaves. No evidence was obtained that one strain of virus protects against infection or injury by a second strain. Six strains, or isolates, covering a range of virulence, are described.

Green peach aphids acquired virus from diseased plants in a feeding period of 10 minutes, and they obtained a maximum virus charge in a feeding period of 6 hours. Aphids reared on diseased plants transmitted virus in a feeding time of 5 minutes; maximum efficiency in transmission was reached in feeding periods of 4 to 6 hours. Virus was retained by aphids for 72 hours but not for 96 hours. The green

peach aphid is very efficient in transmission of the yellows virus; apparently it is much more efficient than the bean aphid and the potato aphid.

The yellows virus appears to be introduced into both parenchyma and phloem by aphid vectors, but introduction into the parenchyma alone delays systemic infection. Virus introduced into beet plants by a vector moved a distance of 10 cm. out of an inoculated leaf in a minimum time of 20 minutes, whereas beet mosaic virus introduced by rubbing required a minimum time of 54 hours to move the same distance. Virus introduced into leaves of *Chenopodium capitatum* by means of vectors moved a distance of 10 cm. out of the inoculated leaf in a minimum time of 40 minutes, whereas virus introduced by rubbing required 4 days to move out of the inoculated leaf. The rapid rates of movement after aphid feeding probably resulted from introduction of the virus directly into the phloem by the vector.

Beet plants apparently do not increase appreciably in resistance to infection with age. Ten green peach aphids per plant have consistently produced high percentages of infection in the greenhouse and on plants of different ages up to 4.5 months in the field.

In chronically diseased beet plants concentration of virus appeared to be higher in leaves just reaching maturity than in old or very young leaves. However, concentration of virus in young leaves of recently infected plants was approximately six times greater than that in leaves of comparable age from chronically diseased plants. In sectored leaves concentration of virus in the yellowed parts was much greater than that in the green areas. In some instances no virus was recovered from the green areas. High initial concentrations of virus in recently infected plants are associated with acute symptoms, and reduced virus concentrations are associated with the chronic stage of the disease in which the plants recover to some degree.

In beet plants with three crowns on the same root system, the virus moved out of the inoculated crown into a defoliated crown and produced symptoms in average times of 12.4 and 14.1 days, respectively, whereas it required 57.3 days to produce symptoms on a noninoculated, nondefoliated shoot of the same plant. The respective periods for movement of mosaic virus and production of symptoms were 9.4, 10.2, and 33.7 days. Mosaic and yellows viruses moved into the noninoculated, nondefoliated shoot in much shorter times than did curly top virus. This finding indicates that part of the movement of yellows and mosaic viruses may have been in the parenchyma, whereas movement of the curly top virus may have been restricted to the phloem. Factors involved in food translocation appear to be important in the movement of all three viruses in the phloem.

Possible control measures consist of (1) spraying to control vectors, (2) destruction of sources of infection, (3) selection of planting dates to avoid infection, and (4) development and use of resistant varieties.

Control of aphid vectors with aphicides appears to be uneconomical in coastal areas of California where aphid populations are high throughout the season, but use of aphicides may be profitable in inland areas where aphid populations drop to low levels in late May or June. Destruction of wild and escaped beets and other sources of infection reduces or delays infection in some areas. December and January

plantings probably are damaged less than later plantings in the Salinas Valley. Plantings made after the middle of May in the San Joaquin and other inland valleys escape infection to some degree, but it is doubtful whether late plantings of this type are often advisable from the standpoint of yield. At present no varieties with high degrees of resistance are available, but it seems probable that varieties considerably more resistant than those now in use may be produced. Development of varieties resistant to yellows appears to offer the greatest hope for eventual control of the disease over much of the area in Western United States, where yellows is now causing the most serious losses.

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