Effects of a Necrosis-Inducing Isolate of Alfalfa Mosaic Virus on Stand Loss in Tomatoes

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ABSTRACT

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Alfalfa mosaic virus (AMV) causes a severe disease of tomatoes that reduces survival of direct-seeded tomato plants in fields adjacent to alfalfa in the Imperial Valley of California. Disease symptoms in tomato plants are stunting, bronzing, and necrosis followed by death. The disease is caused by an unusual strain of AMV that can be transferred mechanically from alfalfa or diseased tomatoes to various host plants, but not to tomatoes. The disease was reproduced in tomato plants by grafting with infected tobacco scions or by inoculating with aphids. The virus was localized in necrotic areas of infected tomato plants, but not always in bronzed or senescent tissues. A previously unreported vector of AMV, the blue alfalfa aphid, and the pea aphid, transmitted the virus experimentally and are probably the principal vectors in nature. Alfalfa was a reservoir of the virus. Patterns of disease in tomato fields indicate the necrosis-inducing strain of AMV spreads from alfalfa into tomatoes with no secondary spread from tomatoes to tomatoes. Results of field and greenhouse trials using commercial tomato cultivars indicated no immunity was present, but suggest that field resistance is associated with lack of infection. Cultivars that were most tolerant in greenhouse trials generally became infected less often during field trials.

In the Imperial Valley, Imperial County, CA, stand reductions occur in tomato fields planted near alfalfa. Losses have been substantial in some fields, and are most common along the margins of fields downwind from and adjacent to alfalfa. Affected tomato plants first become stunted and later develop bronze-colored foliage. Younger leaves and shoot tips become necrotic, and the plants usually die. In addition to the lethal necrotic syndrome, tomato plants with nonlethal, chlorotic, or bright yellow mosaic (calico) symptoms were observed occasionally. Association of these two syndromes with proximity to alfalfa suggests alfalfa mosaic virus (AMV), which is aphid-transmitted, might be involved. However, observation of two distinct disease syndromes implies other agents might be present.

Natural infection of tomatoes with AMV has been reported to occur in France (6), Israel (12), and Australia (3), but not in the United States. Variable reactions of tomato plants experimentally infected with AMV include local lesions, stunting and bronzing of the leaves without death, and mosaic (3,6,7,12). Strains of AMV have been isolated which, under experimental conditions, sometimes cause lethal necrosis in tomato plants. Depending upon the cultivar, however, the symptoms also resemble those caused by other viruses such as CARNA-5 containing strains of cucumber mosaic virus (2,6,7).

This report shows that different strains of AMV present in alfalfa can cause both the necrotic and chlorotic disease syndromes observed in tomatoes in southern California. The mode of transmission and epidemiology of the disease are discussed.

MATERIALS AND METHODS

Virus isolates and inoculations. The following plants were grown in a greenhouse: tobacco (*Nicotiana tabacum* L. 'Xanthi-nc'), pepper (*Capsicum frutescens* L. 'Yolo Wonder'), cowpea (*Vigna unguiculata* [L.] Walp. 'California Blackeye'), pinto bean (*Phaseolus vulgaris* L. 'Pinto'), soybean (*Glycine max* Merr. 'Harosoy'), and *Chenopodium quinoa* Willd. Commercial cultivars of tomatoes (*Lycopersicon esculentum* Mill.) grown in a greenhouse for transmission experiments were: Sunlight, Blazer, Jackpot, Early Bush, Castlemart, Valarie, Duke, Duchess, NCX 3037, NCX 3059 (= Firechief), Full House, Ace, Valley Pride, and Early Pack 7. To test seed transmission of AMV, alfalfa (*Medicago sativa* L. 'Salton') was grown from seeds collected in the Imperial Valley from plants that had been growing for 1 yr.

Isolates of AMV were obtained from diseased field-grown tomatoes or from infected alfalfa plants collected in the Imperial Valley. Isolates inducing necrosis were recovered from tomatoes showing severe disease symptoms or alfalfa by three successive single-lesion transfers from primary leaves of cowpea plants mechanically inoculated with buffered sap, followed by inoculation onto separate healthy tobacco plants. Isolates inducing chlorosis were recovered from tomatoes with chlorotic or bright yellow mosaic (calico) disease symptoms by sap inoculation of tobacco plants. Many isolates from chlorotic tomatoes produced different symptoms in tobacco. All AMV isolates were maintained in tobacco in a greenhouse.

Tissue for mechanical inoculations was ground in a mortar and pestle with inoculation buffer (0.02 M potassium phosphate buffer, pH 7.0, with 0.1% 2-mercaptoethanol, 0.3% sodium diethyldithiocarbamate, and 1% Celite) then rubbed onto leaves of test plants with sterile gauze. Tomatoes were experimentally infected with necrosis-inducing AMV isolates by graft inoculation with tissue taken from tobacco plants mechanically inoculated with the isolates. Rootstocks were prepared from 6- to 8-wk-old tomato plants by severing the stems above the second or third leaf. A piece of tobacco stem with one (or more) axillary bud(s) was then wedge-grafted to the cut end of the tomato. After 10 days, when the graft had set, newly-formed tobacco leaves were mechanically inoculated with a necrosis-inducing AMV isolate. Grafting tomato rootstocks with healthy tobacco scions was more successful than using AMV-infected scions.

Aphid transmission. Blue alfalfa aphids (*Acyrthosiphon kondoi* Shinji and Kondo) reared on lentils (*Lens esculenta* [L.] Moench.), or pea aphids (*A. pisum* [Harris]) reared on broad bean (*Vicia faba* L.) were starved for 1 hr, then placed on leaves of pepper or tobacco infected with a necrosis-inducing isolate of AMV. After an acquisition feeding period from 30 sec to 3 hr, the insects were transferred to tomato seedlings, allowed to feed overnight, then killed with dichlorvos (DDVP). Tomato plants were analyzed by ELISA for AMV after 14 days.

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ELISA system. An enzyme-linked immunosorbent assay system for detecting AMV was developed by using antiserum kindly supplied by R. G. Grogan (University of California, Davis). ELISA reagents were prepared according to the method of Clark and Adams (1), except that DEAE cellulose-purified anti-AMV immunogammaglobulin (anti-AMVIgG) fractions with an $A_{280\ nm}$ greater than 0.8 were pooled and the final concentration adjusted to 1 mg/ml ($A_{280\ nm}$ = 1.4). Wells were scored visually by comparing development of color (indicating presence of AMV) in the sample wells with that of controls.

Double-stranded RNA. Double-stranded RNA (dsRNA) was purified from infected tomato and tobacco plants according to the method of Morris and Dodds (8), and analyzed by gel electrophoresis.

Virus purification. Virus was purified from diseased tomatoes collected in the Imperial Valley or infected greenhouse-grown tobacco plants harvested 10–15 days postinoculation. Tissue was processed essentially according to the method of Van Vloten-Doting (11). Absorbance ratios ($A_{260\ nm}/A_{280\ nm}$) for purified preparations were between 1.62 and 1.77.

RESULTS

Symptoms. Severe disease symptoms in the field were first observed in young tomatoes 10–15 cm tall about 6 wk after direct planting, and continued to appear as late as 2 wk before harvest. Plants affected early in the season became severely stunted and bronze colored. Younger leaves and shoot tips became necrotic and stems, when cut, showed reddish-brown areas of necrotic phloem. Plants usually were girdled by phloem necrosis and died (Fig. 1A and B). Tomatoes affected later in the season sometimes survived, with necrosis and bronzing usually limited to one or more branches. Fruits produced by diseased plants exhibited mosaic or necrotic patches on the skin and often were greatly disfigured by the infection (Fig. 1C).

A second, mild, disease syndrome was observed occasionally in 1980 and 1981, but more frequently in 1982. Affected tomato plants exhibited bright yellow mosaic (calico) symptoms on emerging shoots and/or older leaves, and often, young shoots exhibited extensive interveinal chlorosis. Plants remained robust and no necrosis was observed in leaves or stems. Fruits, however, exhibited necrotic spots, and often were disfigured as in the severe disease syndrome.

Koch's postulates. AMV was always associated with diseased tomato plants. Assays for AMV included use of indicator hosts, serology, negative staining, and analysis of dsRNA from infected or diseased material. In the ELISA system developed for assaying AMV, background from uninfected tissue was routinely less than 0.1 $OD_{405\ nm}$ allowing test results to be scored visually. In initial assays of several hundred diseased field-grown tomato plants, approximately 60% tested positive for AMV. In a subsequent survey of diseased plants, AMV was detected in 17 of 28 samples when tissues were selected that were symptomatic, but not necrotic,

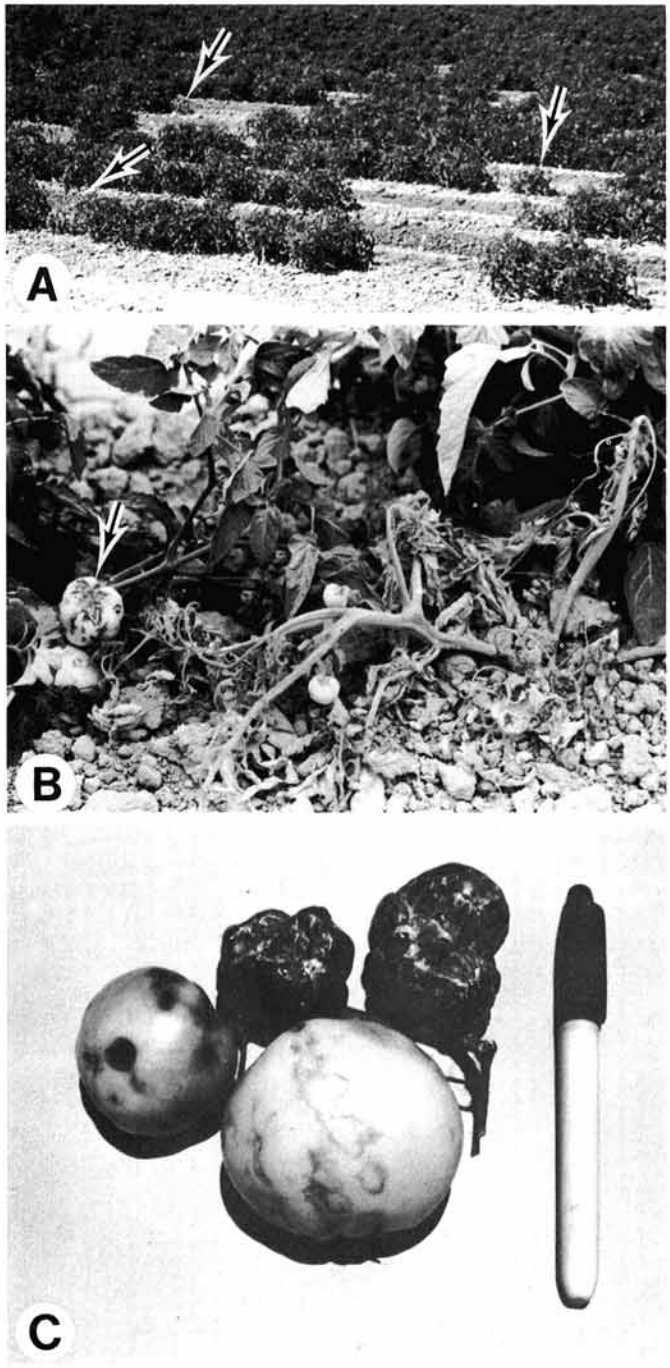

Fig. 1. Symptoms of field-grown tomato plants infected with the necrotic strain of alfalfa mosaic virus. **A,** Bare spots in field are due to death of diseased plants; severely diseased plants indicated by arrows. **B,** A stunted, bronze-colored, and necrotic single severely diseased plant surrounded by healthy neighbors. Arrow points to a necrotic fruit. **C,** Lesions on diseased fruit typical of the severe disease syndrome.

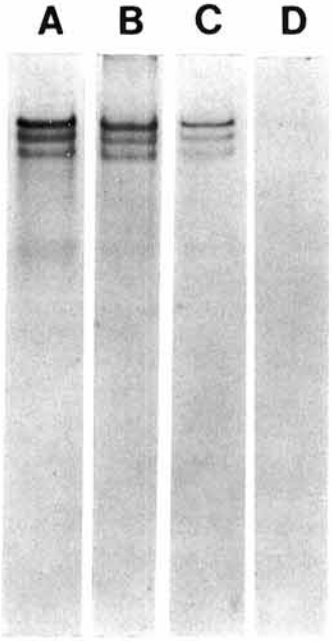

Fig. 2. Polyacrylamide-agarose gel electrophoresis patterns of dsRNAs extracted from: **A,** a severely diseased field-grown tomato plant; **B,** tobacco infected with a known isolate of AMV; **C,** a field-grown tomato plant expressing calico symptoms; and **D,** a healthy field-grown tomato plant.

whereas 9 of 9 samples contained AMV when necrosis-affected tissues were selected. The virus was rarely detected in symptomless field-grown tomato plants. Double-stranded RNAs isolated from either tobacco or tomato plants infected with an AMV isolate from necrotic tomatoes comigrated with those of a known AMV isolate in gel electrophoresis (Fig. 2). Bacilliform particles 18–60 × 18 nm, consistent with those of AMV (4), were purified directly from severely diseased tomatoes. No other virus associated with diseased tomatoes was detected by these procedures.

Tomato plants developed severe disease symptoms when experimentally infected with necrosis-inducing isolates of AMV. Tomato plants grafted with tobacco scions that were later mechanically inoculated with isolates of AMV from necrotic tomato plants were stunted, and exhibited bronzing and necrosis in the phloem, shoot tips, and young leaves 10–20 days after scion inoculation. Grafted tomatoes inoculated with buffer remained symptomless, except for slight necrosis at the graft union. Although individual components of symptoms observed in these experiments were identical to those that occurred in the field, they were often localized, and death, which occurs normally in the field, was rare. However, when six tomato plants infected with a necrosis-inducing isolate of AMV were transferred to a field plot in the Imperial Valley, five developed severe disease symptoms and died; one survived, but developed calico symptoms on the leaves, characteristic of the mild disease syndrome. AMV was detected by ELISA in all of 63 tomatoes that developed symptoms after experimental inoculation with isolates from either necrotic or chlorotic tomato plants, but not in five plants inoculated with buffer.

Characteristics of AMV isolates. Different isolates of AMV exhibited a wide range of symptoms on different hosts. All isolates produced necrotic local lesions on primary leaves of cultivars California Blackeye cowpea and Pinto bean 2 days postinoculation. Chlorotic lesions formed on both inoculated and newly developed leaves of *Chenopodium quinoa*, and a systemic bright yellow mottling was exhibited in developing leaves of Harosoy soybean.

In tobacco, AMV isolates from necrotic tomato plants caused oakleaf or ringspot patterns with thin and broken necrotic margins that were observed on newly developing leaves at about 10 days postinoculation. Symptom expression on developing leaves subsided about 30 days postinoculation, but resumed in leaves that emerged after stems were cut back. Infected peppers exhibited distorted and mottled leaves with mosaic patterns, and symptoms persisted.

Isolates of AMV from chlorotic tomato plants produced different symptoms in different systemic hosts. Infected peppers usually produced distorted leaves with bright yellow mosaic patterns; however, some isolates also caused necrosis of leaf margins. Symptoms in tobacco ranged from calico patterns with distorted leaves that were usually elongated to faint chlorotic mottling. A few isolates infected tobacco without causing symptoms.

Mechanical inoculation of tomatoes. AMV isolates from necrotic tomato plants could not be mechanically transmitted to tomatoes of any age. Mechanical inoculations of 14 commercial cultivars of tomatoes were attempted using a variety of buffers and different sources of inoculum, including purified virions, but these all failed to infect any tomato plant. Cowpea or tobacco, however, were infected in parallel inoculations. Tomatoes could be infected with chlorosis-inducing AMV isolates by mechanical inoculation using the standard inoculation buffer and sap from either infected tomatoes or tobacco. Chlorosis or calico symptoms, as observed in the field, developed about 10–14 days postinoculation.

Localization of necrosis-inducing AMV isolates in tomato plants. The location of AMV within infected tomato plants was determined and correlated with location of symptoms. Tomato plants were inoculated with necrosis-inducing isolates of AMV by grafting with tobacco scions as described previously. At 3 wk postinoculation of tobacco scions, plants were photographed to record location and severity of symptoms. Individual leaflets and 1-cm segments of petioles and stems then were assayed separately by ELISA for AMV (Table 1). AMV was detected in 63 of 64 samples with necrotic tissue, but not always in nearby or adjacent symptomless tissues, even within the same leaf (Fig. 3). The virus was detected in only 29 of 59 (59.5%) samples from bronzed or senescent tissues, but was always localized in the axils of affected leaves.

Virus reservoir. The extent of AMV infection in alfalfa was estimated by using ELISA to analyze samples from Imperial Valley alfalfa fields. The virus was detected in 10 of 11 fields surveyed in 1980 and 1981, although symptoms of infection were rarely observed. In 1980, only one plant was infected of 54 tested from two fields in the first year of growth, whereas 120 plants were infected out of 146 tested (82% infection) from five fields with plantings that were 2–4 yr old. The virus was detected in each of four fields sampled that were adjacent to diseased tomato fields in 1981. AMV

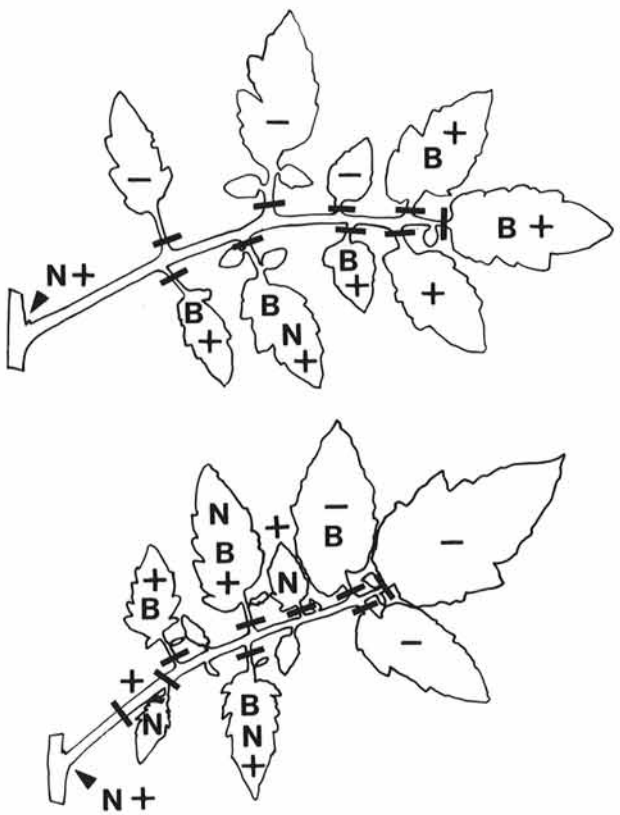

Fig. 3. Location of the necrosis-inducing strain of AMV in experimentally infected tomato leaves. Leaflets of tomato plants, graft-inoculated as described in Materials and Methods, expressed symptoms of bronzing (B), necrosis (N), both necrosis and bronzing (B and N), or were symptomless (no letter). Individual leaflets and stem sections were separated at the location marked by the short bars, and analyzed by ELISA (+ = positive for AMV, − = AMV not detected).

TABLE 1. Occurrence of a necrosis-inducing strain of AMV in individual leaflets of experimentally infected tomato plants[a]

Cultivar	Symptom type		
	Symptomless	Necrotic	Non-necrotic[b]
NCX 3032	5/7	1/1	0/0
NCX 3059	0/9	···	2/5
Full House	6/11	16/16	7/13
Early Bush	23/28	10/11	16/34
Duke	28/65	36/36	4/7

[a] Tomato plants were infected by grafting as described in Materials and Methods. AMV was detected by enzyme-linked immunosorbent assay. Numbers expressed as samples positive for AMV/total samples of the symptom type tested for each cultivar (··· = not tested).

[b] Symptoms other than necrosis included bronzing, yellowing (senescence), leaf distortion, and/or stunting.

was present in seeds and in four of 330 (1.2%) seedlings grown from alfalfa seed collected from plants growing for 1 yr in the Imperial Valley.

AMV was detected, but only rarely, in *Malva neglecta* Wallr. and *Sonchus* spp. L., which are both common weeds in the Imperial Valley.

Spread of AMV into tomato plantings. To determine the pattern of spread of AMV from alfalfa into tomato plantings, disease was measured in two tomato fields adjacent to alfalfa (Fig. 4). In the first field (1981), infection was 29.5% in the first row (45 m from alfalfa), but decreased rapidly to less than 1% at a distance of 200 m from the alfalfa. Infected plants were observed, but were rare, beyond this distance. In the second field (1982), severely diseased plants were counted at 10 wk and again at 17 wk after tomatoes were direct-seeded. When results of both observations were added, total disease was 17.9% in the second row (19.8 m from alfalfa) and decreased to less than 1% at approximately 150 m.

The data were analyzed using linear regression techniques on the natural logs of the infection values against the distance from alfalfa. The values obtained for r^2 were 0.89 for 1981, and 0.53 for the first, and 0.84 for the second (overall) measurements in 1982. Spread of disease in the two tomato fields during a single growing season therefore decreased exponentially with distance from alfalfa. This pattern of disease spread indicates necrosis-inducing strains of AMV move into tomato plantings from adjacent alfalfa with no detectable secondary spread from tomato to tomato.

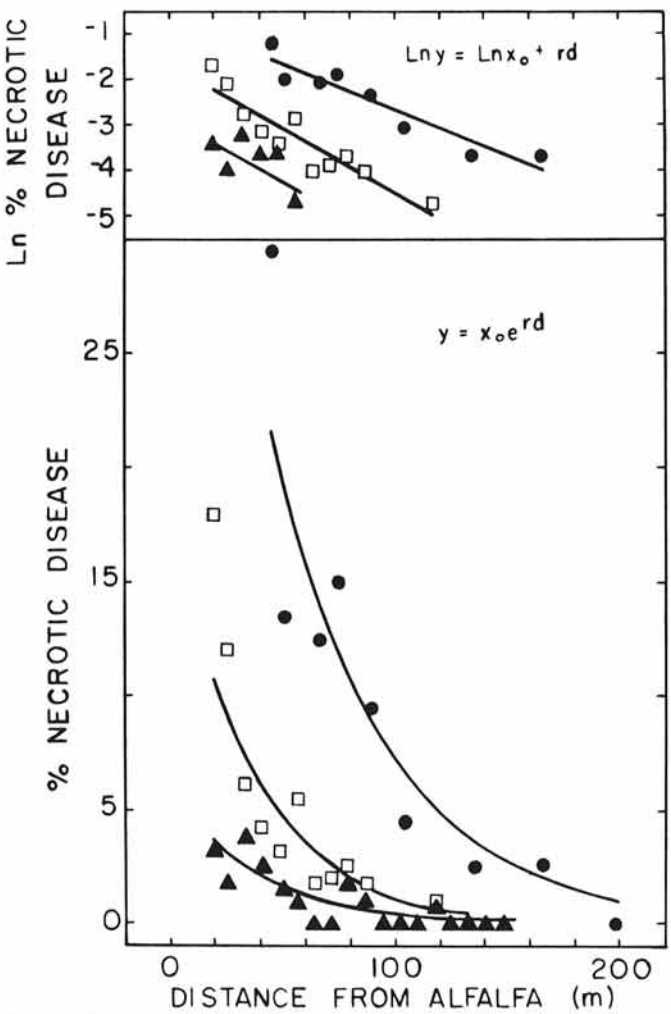

Fig. 4. Relationship between percentage of severely diseased tomato plants and distance from AMV-infected alfalfa. Necrotic tomato plants were counted in one field shortly before harvest in 1981 (●——●). In 1982, necrotic plants in another tomato field were counted at 10 (▲——▲) and 17 (□——□) wk after planting. The linear regression for 1981 is, $\ln y = -0.02x - 0.64$, $r^2 = 0.89$. For the first disease measurement in 1982, $\ln y = -0.027x - 2.85$, $r^2 = 0.53$; for the second (overall) disease measurement, $\ln y = -0.028x - 1.67$, $r^2 = 0.84$.

Aphid transmission. The blue alfalfa aphid and the pea aphid each were able to experimentally transmit necrosis-inducing isolates of AMV to tomato plants in a nonpersistent, stylet-borne manner. In one experiment, five blue alfalfa aphids placed on each of six tomato seedlings after a single probe of infected tissue (~30 sec) resulted in transmission of AMV to each plant. In a similar experiment in which 10–20 aphids per plant and a 1-hr acquisition feeding period were used, blue alfalfa aphids transmitted AMV to 11 of 31 (35%), and pea aphids to five of 30 (16.7%) tomato seedlings. However, the virus was not always efficiently transmitted by aphids. Several experiments were conducted that resulted in no infection of tomato seedlings.

Resistance. Commercial tomato cultivars were screened for field resistance to disease and/or severity of symptom expression from AMV infection. Field trials were conducted in the Imperial Valley in 1981 and 1982 to determine differences in tolerance of tomatoes to natural infection by AMV (Table 2). Results were inconclusive but suggested that cultivar differences do exist. In particular, cultivars Sunlight, Jackpot, and Valarie appeared to be the most tolerant, whereas cultivars GS 1360 and PSR 7979 were most susceptible. Tolerance expressed in field trials resulted from fewer plants becoming infected and not from lack of ability to become infected. In greenhouse experiments, all cultivars tested were experimentally infected with necrosis-inducing isolates of AMV, although slightly different symptoms were usually expressed by

TABLE 2. Tolerance of commercial tomato cultivars to AMV[a]

Cultivar	Trial number		
	One[b]	Two[c]	Three[d]
Sunlight	0.2	0.0	1.0
Blazer	7.05	25.0	1.0
Jackpot	5.8	0.0	0.0
Early Bush	7.05	0.0	1.0
Castlemart II	···	33.0	3.0
Castlemart	7.05	···	···
Valarie	1.2	0.0	0.0
Marquis	···	20.0	···
Duke	···	11.0	1.0
Duchess	···	···	1.0
NCX 3037	17.3	···	···
NCX 3059	···	···	4.0
Full House	···	···	0.0
GS 1360	···	75.0	···
GS 356	···	···	1.0
GS 1379	···	20.0	···
GS 1336	···	50.0	···
PSR 7979	···	85.7	···
FM 52	···	42.8	···
Liberator	···	20.0	···
President	···	25.0	···

[a] Different plot designs and locations were used for each of the three trials conducted in the Imperial Valley, Imperial County, CA. Numbers are percentages of severely diseased (dying or dead) plants of the total for each cultivar in each trial. Results of each trial are not directly comparable (··· = cultivar not tested).

[b] Six rows 20 m long and parallel to an alfalfa field determined by ELISA to be 87% AMV-infected were planted with tomato cultivars obtained from R. Jones, (University of California, Davis). The first row (closest to alfalfa) and second were planted with Jackpot and NCX 3037, respectively. Blazer, Early Bush, and Castlemart were planted in row three, and counted as a single cultivar. Sunlight was planted in rows four, five, and six.

[c] The trial consisted of a single row of 3-m sections of 15 cultivars centered laterally within a large commercial tomato field, parallel to and 21 m from an established alfalfa field infected with AMV. Ten percent of the cultivar Jackpot tomato plants flanking the trial row showed necrotic disease symptoms.

[d] Cultivars were planted in a randomized block of 4.6-m sections in rows parallel to an adjacent alfalfa field determined by ELISA the previous year to be 87% AMV-infected. Castlemart II was planted at the ends of each row. Unfortunately approximately one-half of the alfalfa field was removed 3 wk after the tomatoes were planted.

different cultivars. Cultivars Sunlight and Jackpot generally exhibited the least necrosis and tended to recover, whereas necrosis was most severe in cultivars Early Pack 7 and NCX 3037.

DISCUSSION

Two strains of AMV were isolated from diseased tomato plants grown in the Imperial Valley. These were identified by serology, electron microscopy, and analysis of dsRNA. A necrosis-inducing strain that causes phloem necrosis and death of tomatoes was the most prevalent, and was responsible for losses of tomato plants in the Imperial Valley. The necrosis-inducing strain is unusual because it cannot be transferred mechanically to tomatoes, but easily mechanically infects other hosts. A chlorotic strain that caused nonlethal, calico symptoms was present occasionally.

Virus-induced necrosis appeared to localize the virus, prevent mechanical infection, and induce symptoms not typical of most virus diseases. Presumably, virus introduced into the vascular system by aphids or grafting is able to spread in advance of the necrotic host reaction. Mobility in the phloem and a necrotic host response may explain why this virus was localized in different regions of infected tomato plants, rather than being uniformly distributed. That virus was not necessarily present at the site of symptom expression may also explain why initial screening of diseased tomato plants showed only 60% AMV infection. The stunting and discoloration of the severe disease syndrome probably result from vascular dysfunction due to phloem necrosis. Isolates of AMV from chlorotic tomatoes did not induce necrosis in foliage. However, both forms of AMV caused necrosis in tomato fruits.

We have shown that the pea aphid and the blue alfalfa aphid can each experimentally transmit the virus to tomatoes. This is the first report that the blue alfalfa aphid is able to transmit AMV. These two insects are serious pests of alfalfa throughout California, and are present in peak populations in the Imperial Valley from mid-January to early April (5), coinciding with the time when tomatoes are growing. The new establishment of the blue alfalfa aphid in the Imperial Valley in 1975 (9) parallels increased reports of tomato losses (*unpublished*), suggesting that this vector plays a major role in spreading AMV into tomatoes. Although five other aphid species present in the Imperial Valley are known to transmit AMV, these are probably insignificant in transmitting the virus to tomatoes because high populations do not occur in alfalfa, or coincide with the tomato growing season.

In the Imperial Valley AMV appears to survive in alfalfa. The most prevalent forms of the virus spread rapidly in alfalfa without causing visible symptoms, but cause necrosis in tomatoes. The negative exponential pattern by which the virus spreads from alfalfa to tomatoes was maintained throughout the growing season, indicating there is no secondary spread from tomato to tomato (10). Tomato plants select against necrosis-inducing strains of AMV because infected plants quickly die. In addition, phloem destruction and localization of virus during the course of disease reduce the likelihood that aphids will acquire and transmit the virus. Chlorosis-inducing strains, on the other hand, may arise in tomato by natural mutation and could be selected for by lack of necrosis and host survival. Although tomatoes with the mild disease syndrome occurred too infrequently to quantify, these tended to be in groups within about 10 m of each other, which suggested secondary spread of chlorosis-inducing strains does occur between tomato plants.

No immunity to AMV was found in any tomato cultivar tested. Different cultivars infected experimentally in greenhouse trials showed differences in symptom severity. However, in field trials, infected plants died, so that differences between cultivars were evident only in relative percentages of infection. The reduced amount of death of AMV-infected tomatoes in the greenhouse experiments may be due, in part, to different culture conditions; infected cultivar Sunlight tomato plants showed very little necrosis in the greenhouse, but developed typical lethal necrotic symptoms after transfer to a field in the Imperial Valley. Field resistance of tomatoes, then, is correlated with lack of infection, rather than tolerance to infection. It is interesting that cultivars expressing the mildest symptoms from experimental infection in the greenhouse also became infected less often in field trials.

Future development and use of tolerant tomato cultivars that respond to systemic infection without foliar necrosis and death may permit secondary spread of the virus. The mild strain of AMV causes little foliar growth reduction, but does cause fruit necrosis and appears to spread secondarily in tomato fields. Potentially, given equal amounts of initial inoculum, the mild strain of AMV could cause greater yield losses from diseased fruit than does the necrosis-inducing strain through initial stand reductions.

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