Rate of Spread and Effect of Tomato Ringspot Virus on Red Raspberry in the Field

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ABSTRACT

Tomato ringspot virus moved from plant to plant along rows in two red raspberry fields in Washington State at an annual rate of ca. 2 m. All but 2.9% of new infections in 1970 occurred in plants adjoining infected plants. A low percentage of cucumber seedlings became infected when grown in soil from an infected red raspberry field containing *Xiphinema americanum*. A *Stellaria media* plant growing near infected red raspberries in the field was infected with tomato ringspot virus, but was symptomless. In a Puyallup red raspberry field, healthy plants yielded more than twice the weight of fruit per plant than infected plants. Infected fruits weighed 21% less individually than normal fruits, had only two-thirds as many drupelets per fruit, and were crumbly. Phytopathology 61:1104-1106.

In a survey conducted in 1969, ringspot disease, caused by tomato ringspot virus (TomRSV), was the most common virus disease seen in plantings of red raspberries in Oregon, Washington, and British Columbia (3). In some cultivars, we were able to distinguish between chronically infected plants and those recently infected, and it was evident in two fields that the disease was actively spreading from centers of infection. Since little is known about the ecology of field spread of TomRSV in raspberry, we undertook a 2-year study of the pattern of spread in these two fields. This was supplemented by vector and yield studies.

Tomato ringspot virus causes important losses of yield (4) and fruit quality (2, 6) in certain cultivars. The peach yellow bud strain of TomRSV has been transmitted in the soil by *Xiphinema americanum* Cobb from cucumber to healthy peach, apricot, and plum (11). Previous attempts to transmit TomRSV to herbaceous hosts with nematodes associated with infected red raspberry were unsuccessful (2).

MATERIALS AND METHODS.—Two red raspberry (*Rubus idaeus* L.) fields infected with TomRSV were chosen for this study. Field A was a 7-year-old planting of Puyallup located at Vancouver, Wash., on Lauren sandy loam. The planting was 3.6 acres (1.46 hectares) regularly spaced at 3 × 10 ft (0.9 × 3 m). Field B was an 8-year-old planting of Canby on Puyallup silt loam at Burlington, Wash. It was 1.2 acres (0.5 hectares), with rows 8 ft apart and plants somewhat irregularly spaced 3 ft apart in the row (0.9 × 2.4 m).

Soil samples for nematode population counts were taken from the root zones of infected raspberries with a coring device, 2 × 30 cm. *Xiphinema americanum* were counted after recovery from 28- and 100-mesh screens. Data are presented as counts per liter of soil. For transmission studies, soil from raspberry root zones in Field A was placed in flats, each holding ca. 9 liters of soil. Cucumber (*Cucumis sativus* L. 'National Pickling') seeds were planted in these flats in a greenhouse at Oregon State University and allowed to grow for 29 days. The plants were then harvested and washed, and roots and tops were combined and ground by mortar and pestle with equal volumes of 0.067 M phosphate buffer, pH 6.5, plus 0.1% 2-mercaptoethanol. The resulting sap, mixed with a little Celite, was used to inoculate cotyledons of young cucumbers which were then observed for symptom development.

Identification of the virus isolates obtained during this study was made by testing them in crude cucumber sap on agar gel plates (pH 6.0) with an antiserum against TomRSV (8).

Comparative yield and fruit quality plots were established in Field A. Six replicates of 10 plants each of healthy and infected Puyallup plants were used. Fruit was harvested in seven pickings in July 1969 and weighed. Samples of 25 fruits from each plot were taken during harvest and weighed and disintegrated in a Waring Blendor to obtain the seeds, which were then counted to determine drupelet set.

RESULTS.—*Symptomatology.*—In the spring, TomRSV often caused yellow rings, line patterns, or fine yellow vein chlorosis on leaves of primocanes of some cultivars. These were shock symptoms of recent infections. They disappeared completely in hot weather, and rarely reappeared in chronically infected plants the following season. Chronically infected plants were dwarfed in the spring, foliage was slower to develop than in normal plants, and primocanes had a distinctly darker bronze cast than healthy plants.

Rate of spread.—The state of health of the planting stock that was originally used to set out Fields A and B was not determined. However, it is assumed for each field that several of the plants were infected at the time they were set out, and that these infected plants served as centers for field spread. In both cases, the growers were aware that in their plantings there

were unthrifty areas that had been enlarging for several years. Presumably, there was a higher percentage of infected planting stock in Field A than in Field B.

Plants in Fields A and B were individually rated as healthy, newly infected with shock symptoms, or chronically infected with TomRSV in May 1969 and May 1970. In Field A, 49.1% of the plants were infected with TomRSV in 1969 and 63.6% in 1970. The percentage of plants in the field showing shock symptoms was similar in both years, 12.8 and 11.4, respectively. In Field B, 6.3% of the plants were infected in 1969 and 9.4% in 1970, while the corresponding percentages of plants with shock symptoms were 2.0 and 1.7.

Figure 1-A, B illustrates the patterns of spread of TomRSV in portions of the two fields. Figure 2 is a typical group of chronically infected Puyallup red raspberry plants bounded by plants just becoming infected and merging into healthy plants.

Most new infections developed in plants that were immediately adjoining infected plants (38.6%); or were the first and second plants next to an infected plant (23.6%); or were the first, second, and third plants (14.2%). The weighted average distance that TomRSV spread in 1 year along a row of plants was 6.2 ft (1.9 m). In 12.7% of the cases where adjoining plants in the row could be infected, symptoms failed to occur after 1 year.

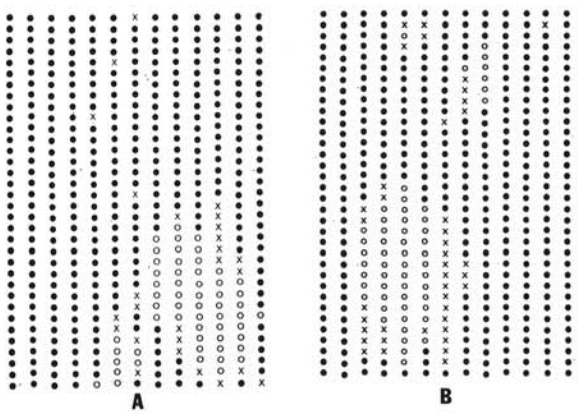

Fig. 1. Pattern of spread of tomato ringspot virus in portions of two red raspberry fields, 1969-1970; **A)** Puyallup cultivar, Vancouver, Wash.; **B)** Canby cultivar, Burlington, Wash. o = Infected when rated May 1969; x = new infections when rated May 1970; • = healthy plant.

Fig. 2. Occurrence of tomato ringspot virus in Puyallup red raspberry, Vancouver, Wash. The dwarfed plants in the center are chronically infected; the taller plants in the foreground are recently infected or healthy.

Detailed maps of spread of TomRSV in Fields A and B were examined for instances of noncontinuous spread (defined as appearance of a new infection with at least one healthy plant adjoining it on both sides in the row and opposite it in the two adjoining rows). In Field A, 15 instances of noncontinuous spread were found in 1970, and in Field B, 3 instances. Over-all, 2.9% of the new infections were noncontinuous.

In order to determine how frequently Puyallup plants near TomRSV-infected plants were infected but symptomless in the spring, 15 groups of three symptomless plants (each group being located adjoining a visibly infected plant) were indexed on an individual plant basis for TomRSV infection on cucumbers. In the case of the first symptomless plants, 10/15 were infected when bioassayed. Among the second plants in line, 7/15 were infected, and among the third plants in line, 3/15 were infected.

Soil transmission.—The population of *X. americanum* in Field A was 119/liter in May 1969, and 207/liter 1 year later. Comparable values for Field B were 218 and 165. Soil obtained from root zones of infected raspberries in Field A in June 1970 was seeded to cucumbers in flats in the greenhouse (ca. 1,800 *X. americanum*/flat). Sap transmissions from these plants 29 days later showed that at least 4/115 (3.5%) of the cucumber plants were infected with virus isolates which reacted with TomRSV antiserum in gel tests.

Weed hosts.—A number of weeds growing in Fields A and B were bioassayed for TomRSV. In 1 of 10 cases, *Stellaria media* (L.) Cyrillo gave an isolate which reacted with TomRSV antiserum. The infected plant was symptomless. No viruses were detected in roots of *Capsella bursa-pastoris* (L.) Medic., *Cerastium* sp., *Cardamine oligosperma* Nutt., *Cirsium* sp., *Epilobium watsonii* Barbey, *Geranium* sp., *Holcus lanatus* L., *Matricaria matricarioides* (Lees.) Porter, or *Senecio vulgaris* L.

Fruit yield and quality.—The results of yield and fruit quality tests in Field A are presented in Table 1. The total fruit yield, weight of 25 fruits, and number of seed per fruit were all significantly greater in healthy plots than in ringspot-infected plots. Most of the fruit from infected plots was crumbly and unmarketable. Six of the 60 infected plants tested were showing shock symptoms, but much of the yield in the infected plots came from these plants. In two plots which had only chronically infected plants, the yield averaged 5.2 kg/10 plants, compared with 13.1 kg/10 plants in two plots in which one-fourth the plants exhibited shock symptoms.

DISCUSSION.—The symptoms of TomRSV infection on red raspberry vary with the cultivar and length of time that plants have been infected. We observed that

TABLE 1. Influence of tomato ringspot virus on yield and fruit quality of Puyallup red raspberry

Treatment	Yield of fruit, kg/ 10 plants[a]	Weight of 25 fruit,[a] g	Number of seed/25 fruit[a]
Healthy	20.2 a	90.0 a	2,041 a
TomRSV	8.5 b	70.9 b	1,376 b

[a] Means with unlike letters in a column differ significantly ($P = .01$).

chronically infected plants were weaker in the centers of large ringspot areas where they had been infected the longest. This variability in symptoms depends on the cultivar and duration of the infection, and may account for the differences in observations on the relationship of TomRSV infection to crumbly fruit (2, 4, 6).

Field spread of TomRSV in red raspberry is predominantly from an infected plant to adjoining healthy plants. The average rate of spread, about 2 m/year, agrees fairly well with rates found for several other nematode-plant virus combinations (1, 5, 7, 10). The intermingling of raspberry roots along and across rows doubtless facilitates transmission. The spread of virus between plants by means of root grafts is not ruled out. The fact that, around the margin of a group of raspberry plants visibly infected with TomRSV, there is a border of plants that are infected but symptomless, does not alter our conclusions with regard to rate of spread. This belt of plants, symptomless but infected, would be expected each year and would constitute a rather constant factor in plotting the rate of spread from symptom occurrence. Any attempts to control local outbreaks of TomRSV in a raspberry field would have to take into account the existence of infected but symptomless plants at the edges of the infected areas.

There were a few clear-cut cases of discontinuous spread in the two fields we studied. We cannot determine if these represent infections by aerial vectors, raspberry pollen (which is an excellent source of TomRSV for sap transmission studies), infection by nematodes transported in the field by soil movement, or nematode acquisition of seed-borne TomRSV from infected weed hosts. These alternatives have recently been reviewed by Taylor & Cadman (9).

Stellaria media from a TomRSV-infected red raspberry field in Maryland was found to be carrying a virus which was probably TomRSV (2). A more thorough study of the relationships of TomRSV and this ubiquitous weed should be undertaken.

Transmission of TomRSV to cucumber in soil from TomRSV-infected red raspberries was demonstrated for the first time in this study. There is still no direct experimental evidence to demonstrate that *X. americanum* can transmit TomRSV to raspberry. However, the dynamics of field spread point strongly to nematodes (very likely *X. americanum*) as the principal vector of this disease in red raspberry.

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