Origin, Cause, Host Range and Spread of Cherry Rasp Leaf Disease in North America

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ABSTRACT

Eleven virus isolates, serologically identical with cherry rasp leaf virus, were recovered from sweet cherry, peach, dandelion (*Taraxacum officinale*), plantain (*Plantago major*), native balsamroot (*Balsamorhiza sagittata*), apple trees inoculated with infected cherry buds, and from the nematode, *Xiphinema americanum*. Rasp leaf disease was induced in cherry cultivars by inoculation with buds from sap-inoculated seedlings of *Prunus mahaleb*, by inoculation of *P. avium* seedlings with infective nematodes, and by implant chips of infected *Gomphrena globosa* plants in California. Natural spread to peach occurred in an orchard and in an experimental plot in California. The herbaceous host ranges of five selected isolates were identical, and 22 of 24 test species became infected. The severity (but not the type) of symptoms varied between isolates and sources, but serological interactions in agar gels showed that all the isolates were closely related or identical, and that they were not related to the viruses which cause Eola rasp leaf and the rasp leaf diseases of Europe. The virus was seed-transmitted in *Chenopodium quinoa* and dandelion. It could be recovered readily from bait plants grown in infested field soil and from *X. americanum* in such soil. Local transmission by nematodes and presence of the virus in balsamroot or other native hosts probably explain the slow spread of the disease, and its occurrence over much of western North America.

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Cherry rasp leaf disease occurs in the western United States and in British Columbia, Canada (1). In California, where its distribution has been studied by Wagnon et al. (16), rasp leaf trees occur in widely separated orchards, but the disease is "usually restricted to a few sweet cherry trees in individual orchards", and it spreads very slowly. Nyland (14) showed that a virus could be sap-transmitted regularly from affected trees; Nyland et al. (15) found that *Chenopodium amaranticolor* Coste et Reyn., *C. quinoa* Willd. and *Gomphrena globosa* L. were susceptible; and that the virus could be transmitted between *Chenopodium* plants by the nematode, *Xiphinema americanum* Cobb. Cherry rasp leaf in the other States and in British Columbia was presumed to be caused by the same virus.

European workers have shown that other Prunus viruses, acting alone or in complexes, can induce enations in cherry foliage (2, 3, 4, 10). In England, cherry rasp leaf disease is usually caused by a combination of prune dwarf virus and raspberry ring spot virus or arabis mosaic virus (Cropley, *personal communication*). Enation symptoms of European rasp leaf diseases are mild and generally scattered throughout the trees, whereas symptoms of North American rasp leaf are very severe and typically restricted to the lower part of the tree.

Eola rasp leaf of sweet cherry trees is a different disease, reported only from Oregon (8, 12). It is characterized by scattered enations, and a drooping habit, while cherry rasp leaf virus induces extremely numerous enations without affecting the habit. Nyland (*unpublished*) found the Eola rasp leaf disease in California, and isolated tomato ring spot virus (TomRSV) from the affected trees of Bing sweet cherry. The isolates were transmitted from cherry to herbaceous hosts and back to cherry and peach in which they produced Eola rasp leaf and peach yellow bud mosaic, respectively.

The purpose of this study was to establish the herbaceous and woody host range of the cherry rasp leaf virus (CRLV); to determine whether isolates from different areas were related or identical; to determine its relationship to other stone fruit viruses; to identify the native host or hosts; and to study its transmission, especially by nematodes.

Purification and characterization of the virus will be reported elsewhere.

MATERIALS AND METHODS.—*Herbaceous hosts.*—The work reported here was conducted at Summerland and Vancouver, British Columbia, and at Davis, California. These locations will be mentioned only where they affect interpretation of the data. CRLV isolates were obtained by grinding affected leaves in 5 ml of buffer and applying the suspension to Carborundum-dusted leaves of *C. quinoa*. In British Columbia, nicotine at either 0.5% or 1% was used for all inoculations. In California, 0.03 M phosphate buffer (pH 7 to 7.2) was used for herbaceous plant inoculations, and the same buffer plus 0.5% nicotine for recovery from stone fruit material. Isolates were routinely maintained in *C. quinoa*, *G. globosa*, or *Solanum sisymbriifolium* Lam. Greenhouse temp were between 18 C and 22 C in British Columbia, and between 22 C and 27 C in California.

Symptoms on herbaceous hots were read about 5, 10 and 20 days after inoculation. Plants that showed no symptoms after 20 days were back-indexed to *C. quinoa* or cowpea, *Vigna sinensis* L. The purity of the isolates was established by inoculating squash, *Cucurbita maxima* Duchesne, *Nicotiana rustica* L., and *S. sisymbriifolium*, all of which are index hosts for other stone fruit viruses (6), but do not react with symptoms when infected with CRLV. CRLV infections in cucumber, *Cucumis sativus* L., and *C. quinoa* could be distinguished from infections of Prunus ringspot and prune dwarf viruses.

Serology.—Antisera were prepared against isolate 1 from California and isolate 7 from British Columbia (Table 1). Both were purified from systemically infected *C. quinoa*. The method used to produce an antiserum against isolate 1 will be reported elsewhere. The citrate-chloroform procedure described previously (5) was used to purify isolate 7. Agar-gel serology tests were made according to the method of Munsi (13), using 0.85% Ionagar No. 2 in 0.85% saline with 0.002% sodium azide on glass microscope slides. Homologous viruses and, to a lesser extent, the other isolates listed in Table 1, were used as antigens. The virus preparations for serological tests were produced in *C. quinoa*, *G. globosa*, *N. clevelandii*, or cucumber. Crude sap from infected plants and sap which had been clarified and concd by ultracentrifugation were used as test antigens. Similar preparations from healthy plants were included as controls.

Nematode transmission.—CRLV in California was transmitted by *X. americanum* between plants of *C. amaranticolor* by Nyland et al. (15). Using similar methods, transmission with *X. americanum* was also attempted from *C. amaranticolor* to five seedlings of *Prunus avium* L.

In British Columbia, soil samples of about 500 cc were taken at several depths in the drip zone of the trees using a 7 × 15-cm bucket auger. Each sample was well mixed and the nematodes were extracted from 100 cc subsamples using the method of McElroy (11). Other 100-cc subsamples were planted with virus-free 1-wk-old cucumber seedlings (cv. 'National Pickling'). Nematodes collected from the extracted portion after being held for 24 h were added to sterile soil which was similarly planted with cucumbers. After 3 wk the plants were removed and their roots were washed free of soil, ground in phosphate buffer, and indexed on *C. quinoa*. Soil samples were considered to contain viruliferous nematodes if the corresponding index on *C. quinoa* resulted in typical CRLV symptoms.

RESULTS.—*Field observations and virus recovery.*—In the Okanagan Valley of British Columbia, infected cherry trees were found in four orchards about 40.2 km (25 miles) from one another. Single trees were infected in three of the orchards, but in the fourth, at Oliver, four adjacent mature trees were infected. CRLV was isolated from each of these trees, from plantain and dandelion leaves and roots in the Oliver orchard, and from balsamroot (*Balsamorhiza sagittata* Nutt.) growing in noncultivated ground near the orchards (Table 1). The virus was not recovered from several grasses nor from 11 chokecherry (*Prunus virginiana* L.) bushes located near orchard areas.

Field observations in California indicate that peach becomes systemically infected with CRLV if infection occurs naturally, presumably via the roots. A few trees in a row of healthy peach trees adjacent to the three inoculated Elberta peach trees referred to above, began to

TABLE 1. Source hosts of cherry rasp leaf virus isolates studied

Isolate number	Source	Symptoms on source host	Location
1.	Peach *Prunus persica* Sieb. and Zucc.	Small enations Severe stunt	Davis, California
2.	Sweet cherry *P. avium* L.	Enations	Deep Creek, B. C.
3.	Sweet cherry	Enations	Summerland, B. C.
4.	Sweet cherry	Enations	Oliver, B. C.
5.	Sweet cherry	Enations	Oyama, B. C.
6.	Sweet cherry	Enations	Corvallis, Oregon
7.	Nematode *Xiphinema americanum* Cobb.		Same orchard as Isolate 4
8.	Dandelion *Taraxacum officinale* Weber	None	Same orchard as Isolate 4
9.	Plantain *Plantago major* L.	None	Same orchard as Isolate 4
10.	Balsamroot *Balsamorhiza sagittata* Nutt.	A few indistinct rings	Non-cultivated area near Oliver, B. C.
11.	Apple (inoculated) *Malus sylvestris* Mill.	Some enations	Summerland, B. C.

show stunted growth and short internodes. The leaf outline changed and lower leaf surfaces were covered with small enations. Sap inoculation to *C. quinoa* showed that the trees were infected with CRLV. Symptoms at first were restricted to the lower parts of the trees, but after three years they appeared in most parts of the trees.

Natural infection in peach occurred in two parts of a young orchard planted on the site of a former cherry orchard near Linden, California, where some trees had been infected with CRLV. Tissues from naturally infected

TABLE 2. Symptoms induced by cherry rasp leaf virus isolate 2 on some herbaceous hosts

Host	Symptoms		Results of back-index
	Inoculated leaves	Systemically-infected parts	
Atriplex hortensis L.	0[a]	0	+
Chenopodium album L.	0	fine mottle	+
C. amaranticolor Coste & Reyn.	0	fine mottle	+
C. murale L.	chlorotic lesions	fine mottle, severe stunt, some epinasty and necrosis	+
C. quinoa Willd.	0	fine band mottle, fine vein clearing of uppermost leaves	+
Cucumis sativus L. cucumber	0 or faint green lesions	fine mottle, slight stunt	+
Cucurbita maxima Duchesne squash	0	0	+[b]
Cyamopsis tetragonoloba (L.) Taub. guar (20 cvs.)	green or brown lesions	0	+[b]
Cyphomandra betacea Sendt	0	0	+
Gomphrena globosa L. Globe amaranth	occasional red lesions	0 fine mottle	+
Nicotiana tabacum L. (3 cvs.)	mostly 0, occasional rings	0	+
N. clevelandii Gray	0	0 or necrosis and leaf distortion	+
N. megalosiphon Heurch & Mueller	necrotic lesions	yellow mottle	+
N. rustica L.	0 occasional rings	0	+
N. sylvestris Spegazzini & Comes	concentric rings	mottle	+
Ocimium basilicum L.	0	0	+
Phaseolus vulgaris L. cv. Pinto	0	0	+
Physalis floridana Rydb.	necrotic lesions	0	+
Sesbania exaltata (Raf.) Cory	brown lesions	0 occasional necrosis	+[b]
Solanum sisymbriifolium Lam.	0	0	+
S. melongena L. eggplant	0	0	–
Spinacea oleracea L. spinach	0	0	–
Verbascum thapsus L. mullein	0		
Vigna sinensis L. cowpea (22 cvs.)	red lesions	0	+[b]

[a] 0 = No symptoms.

[b] Non-inoculated parts remained virus-free unless systemic necrosis occurred.

peach trees were placed in Bing cherry and Elberta peach. Within 2 years, the cherry developed symptoms typical of CRLV near the points of inoculation. The peach trees developed cankers which killed them 5 yr later. Since no rasp leaf symptoms were observed in the inoculated peach trees, the role of CRLV in their death is not clear.

The cherry field symptoms resembled those reported elsewhere (12) and were confined to the lower canopy, except for one of the trees in which the symptoms occurred on all the branches and spurs, but not the current-year shoots. Cherry fruits produced on affected branches had significantly (1% level) shorter pedicels than normal fruit. A total of 50 pedicel length measurements gave a range of 1.9 to 3.3 cm and of 3.0 to 4.5 cm for pedicels from diseased and healthy branches, respectively.

Virus could be recovered readily and consistently from affected parts by sap transmission to *C. quinoa* from the time of bud-break until early summer. After the end of June, recovery became increasingly erratic or impossible. The virus was recovered readily from dormant cherry budwood in two attempts during December and January. From orchard trees of peach, it was recovered during March and April, but not between September and February.

Lott and Keane (9) reported in 1960 that young apple trees inoculated with buds from cherry trees with rasp leaf symptoms developed a few enations after having been pruned back severely. Sap transmission tests were not made. Two of their inoculated apple trees were still available to us and we recovered CRLV from them by sap inoculation to *C. quinoa* (Table 1, isolate 11).

CRLV was not recovered from eight samples obtained from Eola rasp leaf-affected trees in Oregon, but typical CRLV was isolated simultaneously by the same method from branches of a typical cherry rasp leaf stock tree in the Oregon State University collection (Table 1, isolate 6).

Range of herbaceous hosts.—Studies in British Columbia showed that 22 of 24 species and cultivars tested were susceptible to infection with isolates 1, 2, 7, 8, and 10 (Table 2). Only eggplant and spinach were immune. The other isolates were tested on a limited but susceptible range of hosts consisting of *C. amaranticolor*, *C. quinoa*, cowpea, cucumber, *Gomphrena*, guar, *N. tabacum* and *Sesbania exaltata* (Raf.) Cory. All of these developed symptoms similar to those induced by isolates 1, 2, 7, 8, and 10. In most of the 22 susceptible hosts virus symptoms were mild or absent. Cowpea, *Physalis*, guar, and *S. exaltata* generally developed local lesions on the inoculated leaves only, but a few plants of the latter two species developed systemic necrosis. The number of lesions induced by a given inoculum on any one of these four hosts varied from plant to plant, which greatly restricted their usefulness for quantitative assays. When 22 cultivars of cowpea and 20 of guar were inoculated with isolates 2 and 10 and 2 and 11, respectively, the number of resulting lesions varied more between plants of a given cultivar, than between cultivars. Variations in lesion type were associated with the isolate rather than with the test plant: isolate 2 always induced clearly discernible lesions, whereas isolates 10 and 11 induced a few small lesions, or none. *C. quinoa* was approximately 10 times as susceptible as the most sensitive cultivar of cowpea.

Symptom severity on most herbaceous hosts varied according to the time of the year when the tests were conducted. On the most frequently used host, *C. quinoa*, symptoms in the greenhouse in British Columbia were best expressed during spring and fall and were occasionally absent during midsummer even though the greenhouses were no warmer than they were in the winter. During the winter, band mottle symptoms in *C. quinoa* were mild, but the wilting of axillary shoots was a reliable diagnostic symptom. On guar, the interval between

TABLE 3. Recovery of cherry rasp leaf virus from *Xiphinema americanum* collected under cherry and peach trees in British Columbia

Crop	Age (yr)	Virus recovery at depth of			
		0-15 cm	15-30 cm	30-45 cm	45-60 cm
Cherry-A infected	25	+[a] (120)[b]	+ (240)	+(280)	− (100)
Cherry-B infected	25	−[c] (90)	+ (30)	+ (10)	+ (20)
Cherry-C infected	25	+ (40)	+ (110)	+(90)	+ (42)
Cherry-D infected	25	+ (70)	− (13)	− (5)	− (2)
Cherry-E virus-free	25	− (160)	− (50)	− (130)	− (20)
Cherry-F infected (nursery)	1	+ (180)			
Seven peach trees virus-free	2	− (0,0,30, 1,5,5,0)			

[a] + CRLV recovered by bait plants from field soil sample and/or from corresponding sample of washed nematodes.
[b] ( ) number of *X. americanum* in 100 g of soil sample.
[c] − CRLV not recovered by these methods.

inoculation and lesion development was 3-4 days in spring, summer and fall, and up to 13-15 days in midwinter. On *Nicotiana clevelandii*, systemic necrosis and leaf distortion developed at 18-20 C in a heavily shaded greenhouse. The Californian isolate (Table 1, No. 1) when tested in California, infected a limited host range and induced symptoms only in *Chenopodium* spp. No symptoms were noted in *Nicotiana glutinosa* L., *N. rustica*, or in *G. globosa*, although the plants became systemically infected. In repeated tests, the virus was not recovered from inoculated plants of cucumber, cowpea, *Datura stramonium* L., *Dolichos lablab* L., guar, French bean (*Phaseolus vulgaris*), petunia (*Petunia hybrida* Hort.), *S. exaltata*, and periwinkle (*Vinca rosea* L.). When the same isolate was tested under British Columbia conditions, it reacted like the British Columbian isolates.

In British Columbia some isolates could be consistently differentiated on the basis of symptom severity (but not symptom type) in herbaceous hosts: isolate 2 always induced the most severe lesions on *Physalis*, cowpea and guar, whereas isolate 10 induced smaller, more slowly-appearing lesions. On *C. quinoa*, isolates 2 and 10 induced severe and very faint band mottle, respectively. The other isolates induced similar symptoms which were intermediate in intensity between these extremes. On *C. amaranticolor*, *N. tabacum*, and *S. exaltata* the symptoms caused by the different isolates were identical.

In order to observe any variability of symptoms which might be induced by different isolates from one location, we compared individual isolates from the four adjacent infected cherry trees in the Oliver orchard. On *C. quinoa* and *G. globosa*, the characteristic band mottle and the fine vein-clearing of top leaves appeared in all cases, but varied in severity from isolate to isolate. Color intensity of lesions on cowpea varied from dark red to light green and hardly visible. The intensity of the symptoms induced by these four isolates from one location differed almost as widely as did those of the total range of isolates.

Recovery from site of apple orchard.—The orchard at Oliver in which four infected cherry trees were found, and from which isolates 4, 7, 8, and 9 were obtained, was originally planted on newly cultivated land in 1926. In one part of it, apples and peaches were interplanted until 1963 when the peaches were removed. In 1969 the apple trees were removed and the area was used for a cherry tree nursery with seedlings which had been indexed and found free of virus. The seedlings were later budded with virus-indexed sweet cherry cultivars. In 1972, six trees in a 4×6 m area displayed severe symptoms of rasp leaf. The virus was isolated from the affected parts of the trees, from symptomless dandelion and plantain growing among the affected trees, and from soil samples taken near the trees.

Recovery by nematodes.—*X. americanum* was recovered from nearly all the soil samples taken in British Columbia; the populations were much higher under the mature cherry trees and in the young cherry nursery than under some neighboring recently-planted and clean-cultivated peach trees (Table 3). Virus was recovered from nematodes in 12 soil samples taken from under affected orchard and nursery trees. No virus was recovered from nematodes taken from under the indexed virus-free young peach trees or from healthy-appearing cherry trees. Where they occurred, viruliferous nematodes were present in samples from the surface layers to at least a depth of 60 cm. CRLV was recovered once by bait cucumber seedlings to which only 10 nematodes had been added.

In California, four of five mazzard seedlings in containers, which received viruliferous nematodes, showed symptoms of cherry rasp leaf the second year following the exposure. One of the test seedlings died the year following exposure without having shown symptoms. There were no symptoms in five control seedlings that received nonviruliferous nematodes.

Transmission by seed.—Seedlings were raised from four *C. quinoa* plants systemically infected with different CRLV isolates. Assays of individual seedlings showed infection in a ratio of 8/31 from isolate 2, 15/360 from isolate 7, 4/22 from isolate 8, and 1/21 from isolate 10. Similar indexing of dandelion seedlings raised from seed obtained from an infected field plant, showed 4/13 seedlings to be infected. Seed transmission tests in cherry were impossible because seeds from affected parts were dead at harvest time, or failed to germinate.

Transmission by implants.—Chips of tissue from stems of infected *G. globosa* plants were implanted under the bark of yearling mazzard and mahaleb cherry trees. Four of ten mazzard and two of eight mahaleb trees became infected and showed severe enation symptoms on most leaves. The symptoms appeared in the third year following inoculation.

Susceptibility of Prunus cultivars.—In British Columbia, virus-free seedings of several *Prunus* species were sap-inoculated with a partially purified and concd suspension of isolate 2 in 1% nicotine. Two *P. mahaleb* seedlings became infected. Buds from one of these were used to graft-inoculate three cherry trees (cvs. 'Bing', 'Lambert', and 'Van'), three myrobalan plum trees, two apricot trees, and one tree each of almond, peach, plum, and *P. tomentosa* Thunb. The three cherry trees developed leaf symptoms from which the virus was reisolated. The myrobalan trees developed flat limbs and mild unilateral limb-furrowing in a 40-cm section above the inserted buds, but virus could not be reisolated from this affected area, nor from the other inoculated trees.

Observation and back-indexing were continued for 3 yr in the cherry trees. In the 1st and 2nd yr the virus moved 10-25 cm from the point of inoculation. In the 3rd yr it moved along the limbs and into the trunk, still restricted to the lower portions of the three trees. At this stage, pairs of apparently healthy and diseased sections from the same branches were indexed on *C. quinoa*. In eight of ten pairs virus was recovered only from the section with rasp leaf symptoms; one pair showed no virus, and one pair contained virus in both sections. In the 4th yr, the virus advanced still further.

In three Elberta peach trees inoculated in California from a naturally infected peach tree showing systemic symptoms of rasp leaf, cankers developed at the points of inoculation and eventually the limbs were girdled and died. No virus could be recovered by back-indexing from the healthy-looking above ground parts of these trees. The roots were not tested.

Serology.—The antiserum prepared against isolate 1 had a titer of 1:400; that against isolate 7 had a titer of 1:100. Both antisera had a low level of activity against healthy plant components. When crude sap from *C.*

quinoa, *G. globosa*, *N. clevelandii*, or cucumber was used as the antigen in agar-gel serology tests, no specific precipitin lines could be detected. When sap was clarified and concd by ultracentrifugation, single lines of precipitation were evident. The degree of concn required to show a visible line of precipitation varied with the virus isolate, with the growing conditions, and with the host species. In general, sap had to be concd at least 10-fold, and often 50-fold, in order to detect a reaction. All of the isolates of CRLV were serologically related, and the degree of relationship appeared to be close. In a test designed to detect possible serological differences, purified preparations of isolates 1 and 7 were placed alternatively around a depot containing antiserum against isolate 1 and, in a separate test, a depot containing antiserum against isolate 7. A single line of precipitation developed between each antigen-antibody well. The precipitin lines fused without any evidence of a spur reaction, indicating a high degree of serological relationship.

Purified CRLV showed no reaction in tests using a range of antisera prepared against other nematode-borne viruses. Tests included the following antisera: tomato ringspot, cherry leaf roll, strawberry latent ringspot, raspberry ringspot, tomato black ring, peach rosette mosaic, and arabis mosaic.

DISCUSSION.—Our results show that virus isolates obtained from cherry trees infected with CRLV in California and in British Columbia induce similar symptoms on herbaceous hosts and are closely related or identical serologically. Since cherry rasp leaf disease could be reproduced in three cherry cultivars and in mazzard seedlings by inoculation with two isolates that had been characterized in the laboratory and on herbaceous hosts and that seemed to be free of other viruses, we support the conclusion drawn from field studies (14, 15) that cherry rasp leaf disease is caused by the sap-transmissible CRLV. Since the English rasp leaf disease is induced by the synergistic action of two viruses, we suspected that cherry rasp leaf in Western North America might also be due to two viruses. However, all tests indicate that only CRLV can be consistently isolated from cherry rasp leaf affected trees, and that this virus alone induces the disease. The herbaceous host range studies showed that most of the plants tested were susceptible, and that symptom severity can vary considerably within host species such as cowpea and guar. In the absence of facilities for serological work, symptomless infection of squash, the fleeting band mottle on *C. quinoa*, and the extremely mild symptoms on cucumber should be sufficiently diagnostic to distinguish CRLV from necrotic ringspot virus and prune dwarf virus.

The observed differences in herbaceous host range between the California and the British Columbia tests were probably due to environmental influences, such as different greenhouse temp, since the California isolate reacted like local isolates when tested under British Columbia greenhouse conditions. Our stone fruit host range tests were too limited to allow far-reaching conclusions regarding the susceptibility of various cultivars to CRLV. The localized flat limb reaction of myrobalan plum and the lack of virus in the upper nonsymptom-bearing parts of the inoculated cherry trees, served further to confirm that CRLV alone was the agent that induced the rasp symptoms. We have at present no explanation for the variable reaction of peach.

Field transmission of rasp leaf disease seems to occur predominantly by means of *X. americanum*, as shown by the frequent and consistent recovery of CRLV from *X. americanum* collected under affected cherry orchard and nursery trees, by the fact that as few as 10 nematodes per 100 cc of soil were enough to infect bait plants, and by the ease with which cherry seedlings became infected when exposed to viruliferous nematodes under controlled conditions. We were able to show that seed transmission, which is a common characteristic of nematode-transmitted viruses (7), also occurs with CRLV. However, the role which seed transmission and pollen transmission (17) may play in disease spread is not clear. The observed pattern of rasp leaf movement in California is one of slow spread from infected to adjacent healthy trees. It therefore resembles that of other nematode-transmitted cherry virus diseases (7) rather than pollen transmission patterns.

The presence of cherry rasp leaf in a wide area of western North America is surprising in view of the very slow spread and rare occurrence of the disease. As a working hypothesis, we suggest the possibility that CRLV is a virus of the balsamroot and possibly of other indigenous hosts not yet recognized, and that it is spread from these reservoirs into orchards by nematodes.

Our results indicate that cherry rasp leaf disease is of minor economic importance and that occurrences are likely to be local and limited. We suggest that measures to suppress or eliminate nematodes be taken where infested areas are to be planted in cherries. Since CRLV can be recovered by bait plants, it should be possible to determine before planting whether or not an area is infested.

LITERATURE CITED

1. BODINE, E. W., E. D. BLODGETT, and T. B. LOTT. 1951. Raspleaf. Pages 71-80. *in* U.S. Dep. Agric. Handb. 10.
2. CROPLEY, R. 1961. Viruses causing rasp leaf and similar diseases of sweet cherry. Ann. Appl. Biol. 49:530-534.
3. CROPLEY, R. 1964. Further studies on European rasp leaf and leafroll diseases of cherry trees. Ann. Appl. Biol. 53:333-341.
4. DUNEZ, J., R. DELBOS, J. -C. DESVIGNES, C. MARENAUD, J. KUSZALA, and A. VUITTENEZ. 1971. Mise en evidence d'un virus de type ringspot sur Prunus cerasifera. Pages 117-128. *in* Proc. 8th Eur. Symp. Tree Fruit Virus Dis. Bordeaux, 1970.
5. HANSEN, A. J., and R. STACE-SMITH. 1971. Properties of a virus isolated from golden elderberry, Sambucus nigra aurea. Phytopathology 61:1222-1229.
6. KEGLER, H., and J. RICHTER. 1965. The chlorotic-necrotic ringspot virus (CNRV) of sweet cherry. Proc. 6th Eur. Symp. Fruit Tree Virus Dis. Paper No. 35. Belgrad, Yugoslavia.
7. LISTER, R. M. 1960. Transmission of soil-borne viruses through seed. Virology 10:547-549.
8. LIU, K-CH., and T. C. ALLEN. 1965. Serological reaction of virus isolates from cherry with Eola raspleaf. Phytopathology 55:1280-1281.
9. LOTT, T. B., and F. W. L. KEANE. 1960. Leaf enations in

apple inoculated from cherry. Plant Dis. Rep. 44:634-635.
10. MALLACH, N. 1966. Zwetschen als symptomlose Trager von Enationsvirosen der Kirsche. Z. Pflanzenkr. Pflanzenpathol. Pflanzenschutz 73:137-140.
11. MC ELROY, F. D. 1972. Studies on the host range of Xiphinema bakeri and its pathogenicity to raspberry. J. Nematol. 4:16-22.
12. MILBRATH, J. A., and J. E. REYNOLDS. 1961. Tomato ringspot isolated from Eola rasp leaf of cherry in Oregon. Plant Dis. Rep. 45:520-521.
13. MUNSI, W. 1958. Slide gel diffusion precipitin test. Nature (Lond.) 181:1289.
14. NYLAND, G. 1961. Mechanical transmission of the strains of sweet cherry rugose mosaic, mild rusty mottle and rasp leaf viruses. Phytopathology 51:66-67 (Abstr.).
15. NYLAND, G.. B F. LOWNSBERY, S. K. LOWE, and J. F. MITCHELL. 1969. The transmission of cherry rasp leaf virus by Xiphinema americanum. Phytopathology 59:1111-1112.
16. WAGNON, H. K., J. TRAYLOR, H. E. WILLIAMS, and A. C. WEINER. 1968. Investigations of cherry raspleaf disease in California. Plant Dis. Rep. 52:618-622.
17. WILLIAMS, H. E., J. A. TRAYLOR, and H. K. WAGNON. 1963. The infectious nature of pollen from certain virus-infected stone fruit trees. Phytopathology 53:1144 (Abstr.).