Natural Spread of Cherry Rugose Mosaic Disease and Two Prunus Necrotic Ringspot Virus Biotypes in a Central Washington Sweet Cherry Orchard

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ABSTRACT

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The initial appearance and subsequent spread of cherry rugose mosaic (CRM) disease caused by Prunus necrotic ringspot virus (PNRSV) was monitored in a sweet cherry orchard of over 9,000 trees between 1975 and 1986. Between 1975 and 1981, 42 initial disease sites were recognized. Secondary spread occurred at 29 sites despite the fact that diseased trees were usually removed the year that symptoms appeared. Serological tests begun in 1978 detected the presence of another PNRSV biotype that spread at the same rate as the CRM biotype, but that caused no detectable symptoms in cherry trees. These symptomless biotypes were designated HENS (high ELISA, no symptoms). Both CRM and HENS biotypes spread almost exclusively to adjacent trees. The virus spread to an average of only 10% of the adjacent trees each year, and this pattern of spread was not influenced by whether these adjacent trees were pollen compatible or incompatible. Conversely, the probability of CRM spread was greatly reduced by the presence of the HENS biotype, suggesting the natural occurrence of cross-protection between these biotypes. Circumstantial evidence is presented that suggests a relationship between the annual appearance of new CRM sites in the study orchard from 1973 to 1981 and the practice of moving commercial beehives directly from earlier blooming orchards in California to this orchard.

Cherry rugose mosaic (CRM) disease, caused by Prunus necrotic ringspot virus (PNRSV) (12), has been known to occur in Washington sweet cherry (*Prunus avium* L.) orchards for more than 30 years (11). However, the pattern of disease development has changed radically over the past three decades. Before the Washington nursery improvement program was established in 1961, CRM was frequently introduced into orchards through contaminated nursery stock (10). There it spread rapidly as the trees began to flower. By the time orchards reached full bearing capacity (10–15 yr), the disease was often scattered throughout.

Since the early 1960s, most Washington cherry orchards have been planted with virus-certified trees that were free of PNRSV (9). While these orchards appeared to be free from most PNRSV-induced diseases through their early years of development, a dramatic increase in CRM-diseased trees occurred in the mid-1970s (7). Many of these diseased trees appeared in orchards that were initially planted with virus-certified trees (9).

During the late 1970s, circumstantial evidence was presented (8) that showed at least part of the increase in CRM-diseased trees was related to earlier changes in the management of commercial honeybees used by most growers to aid pollination. Tests indicated that most of the Washington-based beehives that returned to Washington after early spring pollination of stone fruit orchards in California contained large amounts of PNRSV antigen in stored pollen (8). This suggested that PNRSV strains (including those that induce CRM) could be introduced repeatedly into "virus-free" orchards through commercial honeybees.

Also recognized during the 1970s was the fact that removal of CRM-diseased trees did not eliminate local spread of the disease, most likely because the disease incited by this pollen-borne virus could not be observed in a newly infected tree before bloom (7). Attempts to locate CRM-diseased trees before bloom during the winter by testing dormant flower buds using enzyme-linked immunosorbent assay (ELISA) showed many orchards contained numerous trees infected with PNRSV strains that caused no recognizable disease symptoms. Since the strains that remained symptomless could not be distinguished serologically from those that induced CRM, ELISA results could not be used in attempts to reduce orchard spread of CRM (9).

There are a few reports that describe the spread of PNRSV in stone fruit orchards; mainly orchards of sour cherry (*P. cerasus* L.) (1–4), prune (*P. domestica* L. subsp. *domestica*) (1), or peach (*P. persicae* (L.) Batsch var. *persicae*) (13). In one case, spread of mild strains of PNRSV was monitored in a sweet cherry orchard (6). However, we are not aware of any report that documents what appears to be two distinct stages of CRM spread in sweet cherry orchards: initial disease occurrence, and subsequent localized spread. Furthermore, we are not aware of a comparable situation in other stone fruits where two serologically identical but biologically different strains (biotypes) occur and spread in the same orchard but only one biotype induces an economically important disease.

This report records the recognition and patterns of spread of two PNRSV biotypes in a single sweet cherry orchard over an 11-year period and discusses some of the implications of these findings.

MATERIALS AND METHODS

Survey orchard. The orchard where this study was conducted is representative of many sweet cherry orchards throughout central Washington. It was planted in 1962 and initially contained 9,423 trees located approximately 6 m apart in rows spaced 6 m apart. Many of the trees were known to be propagated from virus-free sources. However, records are unclear as to whether or not the trees were virus-certified when planted. Although a few scattered trees were removed during the first 12 years for various reasons, no

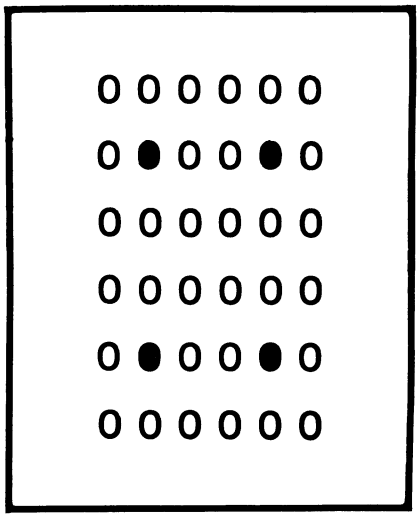

Fig. 1. Typical arrangement of cultivars in a central Washington sweet cherry (*Prunus avium* L.) orchard. (open circle = Bing; closed circle = Black Republican).

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virus-diseased trees of any type were recognized in the orchard until rugose mosaic disease was confirmed in 1975.

Orchard management practices. Like most eastern Washington orchards, approximately 88% of the original trees were Bing, a self-infertile, black-fruited cultivar, which is normally harvested for fresh market in early June. Black Republican trees, which are normally harvested later for processing, were located at every third tree space in every third row as a compatible pollen source for the Bing trees. This arrangement assured that one pollinizer tree was located adjacent to every Bing tree (Fig. 1).

Beginning in 1972, and annually thereafter, 10 rented beehives were placed at each of 10 different locations scattered throughout the orchard (100 hives total) to aid pollination during the short bloom periods. Each year the rental bees were used to pollinate stone fruit trees in California before their use in Washington.

Whole orchard surveys. Although symptoms of CRM include leaf distortion and reduced shoot growth (9), the disease is of concern to growers because ripening of Bing fruit is delayed anywhere from a few days to several weeks. Consequently, every year just before harvest, the grower examined every tree in the orchard for uniform fruit maturity and unusual leaf symptoms. Any diseased tree was flagged and most were removed during that same fall or winter. The following year, a young tree of the appropriate cultivar was replanted in the open space.

Independent of the grower's activities, we examined every orchard tree for CRM before harvest in 1977, 1978, 1981, 1982, and 1985. Results of our observations are presented below as whole orchard survey data. Data for the intervening years are based on the grower's tree removal records and on the age of replanted trees.

Although the grower provided us with his annual tree removal list and was generally familiar with our observations, he did not use our visual survey data or our serological indexing results in any management decisions that involved tree removal. As a consequence, the data presented below on virus distribution and rate and patterns of spread were probably not influenced by our research.

Detailed study areas. In 1976 we selected six rectangular areas within the orchard where individual trees were examined at least twice each year. Each area included between 66 and 121 trees. Areas 1–5 were selected initially because they consisted of one or more rugose mosaic-diseased trees surrounded by many apparently healthy trees. Area 6 consisted of 121 apparently healthy trees, all of which were located more than 11 tree spaces from the nearest diseased tree in 1976.

Beginning in the winter of 1978–1979, and annually thereafter, every tree in each of the six study areas was indexed by ELISA for PNRSV at least once before bloom.

ELISA conditions. All conditions for ELISA were the same as those described earlier (7). During the winter months, dormant flower buds were collected from four main scaffold limbs of each tree and tested for PNRSV. Some confirmatory tests were made using flower, leaf, or fruit tissues collected during the spring and early summer.

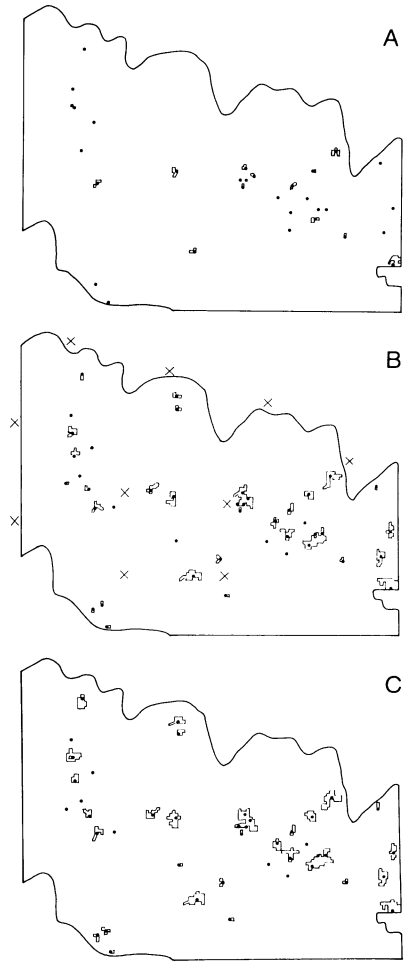

Fig. 2. Distribution of cherry rugose mosaic-diseased trees in a central Washington sweet cherry orchard in **(A)** 1977, **(B)** 1981, and **(C)** 1985. Solid circle = initial location of a rugose mosaic-diseased tree. Open rectangles = additional diseased trees at a given site. X = location of 10 rental beehives.

Table 1. Incidence of cherry rugose mosaic-diseased trees between 1977 and 1985 in a 9,423-tree central Washington sweet cherry orchard

Year surveyed	Trees exhibiting disease symptoms[a]	Cumulative no. diseased trees removed[b]	Cumulative no. diseased trees
1977	20	44[c]	64
1978	37	64	101
1981	45	186[c]	231
1982	19	231	250
1985	25	280[c]	305

[a] Observed during June just before harvest.
[b] Cumulative number of diseased trees previously removed.
[c] Determined from the grower's tree removal records.

Table 2. Relationship between the age of all cherry rugose mosaic (CRM) disease sites and the number of diseased trees recorded at those sites in 1985 within a 9,423-tree central Washington sweet cherry orchard

Year surveyed	Number new disease sites	CRM disease site age (1985)	Average no. diseased trees per site (1985)
1973	1	12	21
1974	2	11	15
1975	7	10	11
1976	9	9	8
1977	11	8	8
1978	8	7	5
1979	2	6	4
1980	1	5	4
1981	1	4	2

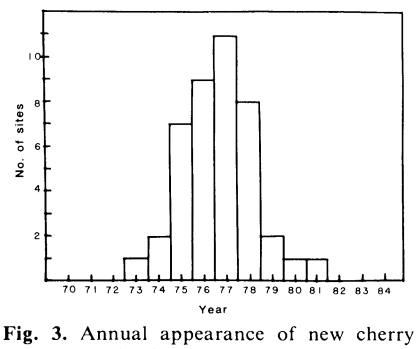

Fig. 3. Annual appearance of new cherry rugose mosaic disease sites (no. of sites) in a 9,423-tree central Washington sweet cherry orchard.

RESULTS

Occurrence and distribution of initial disease sites. During our first full orchard survey in 1977, we identified 20 rugose mosaic-diseased trees (Table 1). Based on the grower's tree removal records and the age and distribution of replanted trees, it was apparent that 44 additional trees had been removed before 1977 because of CRM.

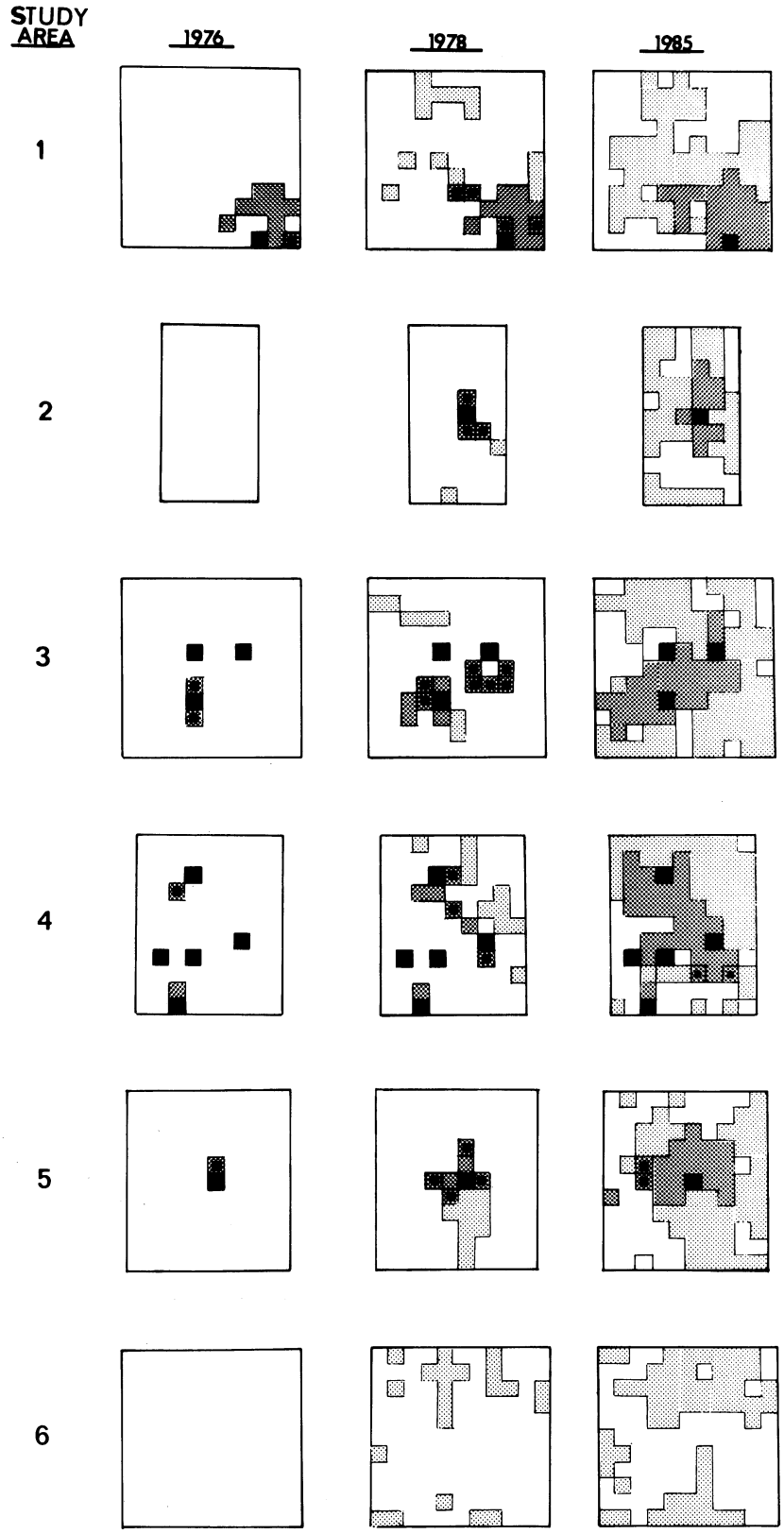

Fig. 4. Diagramatic representation of six study areas within a central Washington sweet cherry orchard depicting the locations in 1976, 1978, and 1985 of healthy trees and trees infected with Prunus necrotic ringspot virus biotypes CRM (cherry rugose mosaic) and HENS (high ELISA, no symptoms). Black square = initial CRM-diseased tree, dark shaded area = CRM-diseased tree removed, dark shaded area with black center = CRM-diseased tree, light shaded area = HENS infected tree, and white area = healthy tree.

These 64 diseased trees were situated in 30 different sites scattered throughout the orchard (Fig. 2A). Seventeen of these sites consisted of a single rugose mosaic-diseased tree. Eleven of these single tree sites were recognized for the first time in 1977. The remaining 13 sites consisted of two or more diseased trees that appeared to have developed around single diseased trees.

As far as we could estimate from the grower's records, the first tree in the orchard to express CRM symptoms did so in 1973 and was removed the following year. After that, the number of new disease sites that appeared each year increased until 1977, when 11 new sites were recorded (Fig. 3). Beginning in 1978 the number of new disease sites found each year decreased until 1981 when the last new site was recorded.

The 42 disease sites that were recognized by 1981 were scattered across the orchard with no apparent relationship to the edges of the orchard or to locations where beehives were placed each year (Fig. 2B).

There were minor, but consistent, differences in symptomatology between some disease sites, indicating that some variation existed among the biotypes studied at the different sites.

Secondary spread of rugose mosaic disease. It is obvious from Figure 2C that secondary spread occurred at 35 of the 42 disease sites, even though the initial diseased trees were removed within 1–2 yr of their appearance. The total number of diseased trees found at a given site in 1985 ranged from 1 to 20. The average number of diseased trees recorded at each site was directly related to site age (Table 2).

By 1985, a total of 305 rugose mosaic-diseased trees had been recorded in the orchard (Table 1), with 263 (86%) apparently the result of localized secondary spread. Of the 263 diseased trees, 251 (95%) were, at one time, symptomless trees located adjacent to a diseased tree. The remaining 12 (5%) were at one time located no more than one tree space from a diseased tree.

Occurrence of two PNRSV biotypes within the study areas. When we began serological tests in the six study areas during the 1978–1979 winter, we had already identified a total of 50 rugose mosaic-diseased trees in five of the six study areas. Although 29 of these diseased trees had been removed by 1978, their locations and the locations of the 21 remaining diseased trees formed irregularly shaped clusters in study areas 1–5 (Fig. 4). As expected, PNRSV was detected by ELISA in dormant bud tissues from all 21 existing diseased trees. Unexpectedly, however, the same tests revealed that 65 apparently healthy trees were also infected with PNRSV. These

infected but symptomless trees were found scattered throughout all six study areas (Fig. 4). While these symptomless trees were not necessarily located near a diseased tree, it seemed logical at the time to assume that they had recently become infected with disease-causing PNRSV isolates, and would sooner or later express symptoms. However, all of these originally infected but symptomless trees remained symptomless throughout the 1979 growing season, and each has remained symptomless every year since.

In separate studies, we have isolated PNRSV from several of the diseased and symptomless trees and have established that several of these isolates are serologically similar, but biologically distinct, PNRSV variants (biotypes) that occur in this and other Washington orchards (5,9,10). The symptomless biotype was designated HENS (high ELISA, no symptoms) (5).

Incidence and spread of PNRSV biotypes within the study areas. Within the six study areas, the total number of trees infected with each PNRSV biotype was similar in 1978 (Fig. 5). However, over the next 8 years we detected two to three times more HENS-infected trees than CRM trees. By 1985, nearly 45% of the 633 trees in the six study areas were infected with the HENS biotype, whereas only 22% of the 512 trees in study areas 1–5 had exhibited CRM. This difference in the accumulated number of tree locations affected by these biotypes was most likely influenced by annual roguing of CRM-diseased trees.

Most of the study area trees that became infected with either PNRSV biotype were located adjacent to a tree already infected with the same biotype (Table 3). For example, of the 161 healthy trees that became infected with the HENS biotype between 1978 and 1985, situated such that the proximity of a previously infected tree could be discerned, 145 (90%) were located next to a previously HENS-infected tree. Similarly, 95% of the trees that developed CRM during this period were located adjacent to a diseased tree.

Although, as noted above, many more HENS-infected than CRM-infected (diseased) trees were observed in the years after 1978, the actual rates of spread for both biotypes from infected to adjacent trees were nearly equal. An average of 10% of the healthy trees adjacent to infected trees became infected each year during the period from 1978 to 1985 (Table 3). Since both biotypes appeared to spread at similar rates from known sources of infection, the reduced incidence of CRM relative to HENS biotypes was most likely due to the annual removal of diseased trees and, thus, of inoculum sources of the CRM biotype. Although the rate of spread to adjacent trees averaged 10% per year, it varied annually from 2 to 18% (Table 4).

Rugose mosaic disease rarely developed in HENS-infected trees. Between 1978 and 1985 only 3, or 0.6%, of 508 (represents the accumulated amount of annual totals) HENS-infected trees located adjacent to rugose-diseased trees became diseased (Table 3).

Cultivar susceptibility. The ratio of Bing to Black Republican trees that were infected annually with either PNRSV biotype was similar to the ratio of trees in the population (8.0) (Table 5). Furthermore, the virus appeared to move readily from infected trees to adjacent trees of the same cultivar. For example, at 22 of 25 locations where the initial rugose mosaic-diseased tree was Bing, the next nearby tree to exhibit symptoms was also Bing.

DISCUSSION

Initial sites of CRM seemed to appear more or less at random throughout the orchard during the 1970s, when it was common practice to move beehives directly from California stone fruit orchards into blooming cherry orchards in Washington. During that period, it was not unusual for beehives to be removed from a California orchard around 5 p.m. and be placed in a Washington orchard early the following morning (8). Viable pollen contaminated with infectious PNRSV could be

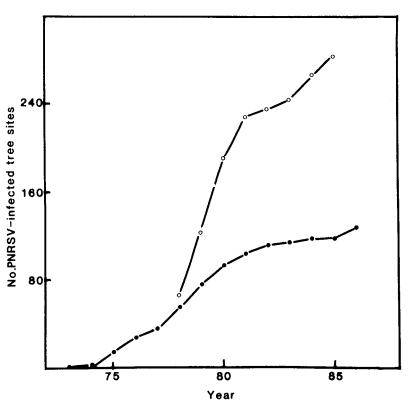

Fig. 5. Incidence of two PNRSV biotypes (closed circle = CRM [cherry rugose mosaic] and open circle = HENS [high ELISA, no symptoms]) in six study areas within a central Washington sweet cherry orchard (represents a combined total of 633 trees).

Table 3. Influence of a tree's virus status and its distance from an infection source on the probability that it would become infected with either of two *Prunus* necrotic ringspot virus (PNRSV) biotypes within six study areas in a central Washington sweet cherry orchard[a]

Condition of recipient tree	Tree spaces from infected tree (no.)	Biotype of nearest infected tree: CRM[b] Trees observed (no.)[c]	CRM[b] Trees infected No.[c]	CRM[b] Trees infected %	HENS[b] Trees observed (no.)[c]	HENS[b] Trees infected No.[c]	HENS[b] Trees infected %
Healthy	1	628	63	10.0	1,347	145	10.8
	2	519	3	0.6	332	10	3.0
	3	1,397	0	0.0	138	6	4.3
HENS-infected	1	508	3	0.6	...	...	...
	2	400	0	0.0	...	...	...
	3	642	0	0.0	...	...	...

[a] Summarizes exposures between 1978 and 1985.
[b] PNRSV biotypes: CRM = cherry rugose mosaic; HENS = high ELISA, no symptoms.
[c] Accumulated amount of annual totals for the years from 1978 through 1985.

Table 4. Annual incidence of new infections of the CRM and HENS biotypes[a] of *Prunus* necrotic ringspot virus in trees located adjacent to previously infected trees within the study areas[b] of a central Washington sweet cherry orchard

Year surveyed	HENS: Healthy trees bordering infected trees No.	HENS: No. infected the next year	HENS %	CRM: Healthy trees bordering infected trees No.	CRM: No. infected the next year	CRM %
1978	167	26	15.6	118	16	13.6
1979	222	40	18.0	116	14	12.1
1980	220	33	15.0	86	11	12.8
1981	188	5	2.7	75	7	9.3
1982	187	9	4.8	67	2	3.0
1983	189	28	14.8	63	4	6.3
1984	174	4	2.3	54	1	1.9
1985	...	*[c]	*	49	8	16.3
Total	1,347	145	10.8	628	63	10.0

[a] CRM = cherry rugose mosaic; HENS = high ELISA, no symptoms.
[b] Represents a combined total of 633 trees. However, the data in this table represent only those trees whose proximity to the nearest infected tree could be discerned.
[c] * = Not observed.

detected on the bodies of bees that emerged from such hives (G. I. Mink, *unpublished data*). By 1978, we discussed with the grower the possible role that beehives might play in introducing CRM into the orchard. The following year we recommended to the beekeepers' associations of both Washington and Oregon and to cherry growers of both states that beehives from California should be held without access to flowering stone fruit trees for at least 2 weeks before being moved into local cherry orchards. This recommendation soon became common practice throughout Washington. While the timing of these events may be fortuitous, our survey data demonstrate that no new disease sites appeared in the study orchard after the change in bee management practices became general.

To provide additional evidence toward the hypothesis that rental bees from California are a source of initial infection in Washington orchards, we have visually and serologically monitored a 500-tree Washington sweet cherry orchard of similar age and origin since 1963. This small orchard is located approximately 5 km from the orchard discussed above. However, California bees have never been used in the smaller orchard. No case of PNRSV infection has yet been found in this orchard in 24 years.

Because the HENS biotype of PNRSV was not recognized until 1980–1981 and HENS-infected trees cannot be identified without either ELISA or host range data, we cannot determine accurately when or how this biotype was introduced into the study orchard. We postulate, however, that it was introduced in a manner similar to that of the CRM biotype, and probably during the same period (after 1971).

While the circumstantial evidence appears strong that the CRM biotype of PNRSV was repeatedly introduced into the study orchard through honeybees, our orchard maps fail to show any obvious relationship between the location of the 42 initial disease sites that appeared between 1973 and 1981 and the annual placement of the beehives during that period. This apparent lack of relationship might be related to initial foraging patterns of honeybees.

The patterns and rates of secondary spread of both PNRSV biotypes were similar, if not identical. Spread occurred at equal rates and almost exclusively to adjacent trees. This pattern of secondary spread of PNRSV was also reported to occur in sour cherry orchards in eastern North America (3,4).

Although annual roguing of CRM-diseased trees did not eliminate the disease, it appears to have been partially effective in reducing disease incidence. In 1978, the accumulated numbers of infected trees of both the HENS biotypes (symptomless) and the CRM biotype (disease) of PNRSV were approximately equal in the study areas. Despite observations suggesting that both biotypes spread locally at equal rates (to approximately 10% of the adjacent trees annually), by 1985 the accumulated incidence of the HENS biotype was nearly double that of the CRM biotype. Most likely, this differential was due to the annual removal of CRM inoculum sources.

Our observations provide some insight into three aspects of secondary PNRSV spread within a commercial sweet cherry orchard. First, considering the fact that hundreds of honeybees repeatedly visit thousands of blossoms on adjacent healthy and PNRSV-infected trees during the bloom period and only an average of 10% of the adjacent trees become infected each year, the rate of PNRSV transmission through pollen to a mature tree per bee visit must, indeed, be very rare. Second, the probability of a healthy tree becoming infected appears to be unrelated to pollen compatibility factors, suggesting that fertilization is not required for transmission to occur. Third, while the rate of spread of both PNRSV biotypes appeared to vary from year to year, the pattern of this variability was similar for both biotypes. This third observation suggests that some undefined environmental factors can affect the efficiency of PNRSV transmission.

As we have previously reported (5), and as the data presented in Table 3 substantiate, HENS-infected trees located adjacent to CRM-diseased trees rarely develop disease symptoms. Only three such cases were recorded over an eight-year period. The fact that over 99% of all HENS-infected trees remain symptomless, even when located near diseased trees, strongly suggests that the HENS biotype provides natural protection to sweet cherry trees against either subsequent infection by CRM-inducing biotypes or against disease expression if such infections occur. Studies are now in progress to determine if such protection can be adapted to reduce the economic impact of CRM disease in Washington state.

Table 5. Annual ratio of Bing to Black Republican (cultivars of *Prunus avium* L., sweet cherry) trees that became infected with either of two biotypes of *Prunus* necrotic ringspot virus within six study areas[a] of a central Washington orchard (population ratio of these respective cultivars within the orchard was 8)

Year surveyed	CRM biotype[b]			HENS biotype[b]		
	Bing	Black Republican	Ratio	Bing	Black Republican	Ratio
1978	46[c]	7	6.6	61	5	12.2
1979	63	9	7.0	109	12	9.2
1980	80	11	7.3	175	15	11.7
1981	92	11	8.4	188	23	8.2
1982	97	13	7.5	195	23	8.5
1983	100	13	7.7	201	23	8.7
1984	104	13	8.0	233	26	9.0
1985	105	13	8.1	252	27	9.3

[a] These study areas contained a combined total of 633 trees.
[b] CRM = cherry rugose mosaic; HENS = high ELISA, no symptoms.
[c] Number of infected trees.

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