Influence of Prunus Necrotic Ringspot Virus on Growth, Productivity, and Longevity of Peach Trees

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ABSTRACT

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Peach trees in experimental plantings at Byron and Fort Valley, GA, were assayed for Prunus necrotic ringspot virus (PNRSV) each year for 3 yr. Three orchards consisted of two peach cultivars (Redhaven and either Loring or Redskin) on seven or eight peach seedling rootstocks, whereas a fourth orchard consisted of only Redhaven scion on Lovell and Siberian C rootstocks. PNRSV was detected by enzyme-linked immunosorbent assay using antiserum prepared against PNRSV-G. Initially, when the orchards were 5–9 yr old, incidence of positive tests ranged from 26 to 63%. The average annual increase in positive assays for these orchards ranged from 3.6 to 17.4%. Trees that tested positive for the virus tended to have a smaller trunk circumference than those that tested negative, but statistical differences in growth during 3 yr were detected in only one orchard. Bark splitting was associated with positive tests in three out of eight cases in which data were collected. Yield of infected trees was reduced by 8.2–47.3% ($P \leq 0.05$) in three out of nine cases, and fruit maturity was affected depending on rootstock. Defoliation, flower bud density, flower and leaf emergence, fruit size, and peach tree gummosis caused by *Botryosphaeria dothidea* were generally not affected by the virus. Although PNRSV has been suspected as a possible factor in the development of peach tree short life, no relationship was found between PNRSV and factors associated with peach tree short life such as cambial browning or bacterial canker caused by *Pseudomonas syringae* pv. *syringae*. Furthermore, PNRSV did not contribute to tree mortality, which in all orchards was the highest (7.1–30.8%) for Redhaven trees. Interactions between virus and rootstock were frequently detected, indicating that the use of virus-free propagation material in rootstock evaluation programs is important.

Prunus necrotic ringspot virus (PNRSV) is common in most areas of the world where stone fruit are cultivated. It exists as a number of strains that cause a diversity of symptoms on many hosts (16,17). In areas of the United States where peaches (*Prunus persica* (L.) Batsch) are grown, symptoms associated with infection by the virus include foliar chlorotic rings, necrotic spots or deformation, and bark necrosis, pitting, splitting, or girdling (20). PNRSV is transmitted in seed and pollen and may be distributed in propagating materials (4,13,20). It is sometimes found in combination with other stone fruit viruses, particularly prune dwarf virus (PDV) (18,22).

The influence of PNRSV on growth and yield of cherry and prune trees in the United States has been well documented (7,8,11,14,15,21). In peach trees, this virus is reported to have a negative effect on growth and yield in California (6,23,26), France (25), and Japan (34). Based on observations of peach in California (2), PNRSV contributes to increased sunburn, chronic dieback, shorter tree life, and predisposition to *Cytospora* and *Armillaria* infections. Recently, the presence of PNRSV was associated with a slow decline of peach trees in central Georgia (32).

PNRSV has been suspected as a possible contributing or predisposing factor in the peach tree short life (PTSL) syndrome, which is a major cause of peach tree death in the southeastern United States. PTSL is characterized by sudden collapse of new growth and premature death of peach trees, usually in late winter and spring (24). Cold injury and/or bacterial canker caused by *Pseudomonas syringae* pv. *syringae* van Hall have generally been associated with PTSL death of trees (5,24,31,33). The purpose of our study was to investigate growth, fruiting, and factors associated with PTSL as they relate to the presence of PNRSV in peach trees.

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MATERIALS AND METHODS

Peach orchards. Four plantings were studied. Orchard I was planted at Byron in 1976 as part of the S-97 Regional Peach Rootstock project (5). Cultivars Redhaven and Loring were budded on peach rootstocks Halford, Harrow 208, Lovell, NA-8, Nemaguard, Siberian C, NC NRL-4, and NC 152-AI-2. Each scion-rootstock combination was replicated four times in six-tree plots. Similarly, orchards IIA and IIB were established in 1981 at Byron and Fort Valley, respectively, as part of the S-97 project. In both plantings, cultivars Redhaven and Redskin were growing on Halford VF, Lovell VF, Nemaguard, Tennessee Natural Redleaf, S-60, NC 503I-4, and NC 88I-2AC-2. Four replicates of four trees each were used. Orchard III was established at Fort Valley in 1980 with the cultivar Redhaven grown on Lovell and Siberian C rootstocks in four-tree plots, 24 plots per rootstock. In each orchard, replicate plots were arranged in a randomized complete block design.

Virus assays. PNRSV assays for orchard I were performed each year from 1984 to 1986 and for the other three orchards from 1985 to 1987. Additionally, all four orchards were assayed for PDV in 1986. From each tree, one growing shoot tip was removed from three sides or three scaffold limbs and stored for no more than 48 hr at 5 C until assayed. The samples were assayed using the enzyme-linked immunosorbent assay (ELISA) procedure described by Clark and Adams (3). Antisera were obtained from the American Type Culture Collection, Rockville, MD. PNRSV antiserum was PVAS-22 prepared against Fulton's isolate G and PDV antiserum was PVAS-290 prepared against the E. Halk 876 isolate. A composite of leaf tissue from shoot tips of each tree was ground in 0.1 M sodium phosphate buffer, pH 7.0, at 1:20 (w/v) ratio. Immunoglobulin and conjugated immunoglobulin were used at 1 μg/ml. Incubation periods were 4 hr for coating and conjugate fixation and 12 hr at 6 C for the sap-filled plates. Each sample was tested in adjacent duplicate wells. ELISA plates included controls (from corresponding peach tissues) known to produce positive or negative reactions. Results were recorded using a Dynatech MR 600 microplate reader (Dynatech Instruments, Inc., Torrence, CA).

Vegetative growth. Trunk circumferences were measured 30 cm above ground in December of each year, and increases in trunk girth were calculated for a 3-yr period. Degree of vegetative budbreak was rated when the first tree in the orchard reached full bloom, using a 1–9 scale adopted from the S-97 regional project (5), where 1 = fully dormant buds

and 9 = leaf shoots 7.5-cm long. Rootstock suckers were counted in late September. Autumn defoliation was rated in November when the first tree in the planting was completely defoliated. A 1–9 scale was adopted from the S-97 project, where 1 = less than 20% and 9 = 90–100% defoliation.

Flower and fruit production. Flower bud density was determined in January from counts of live buds per 10 cm of wood, using terminal sections of five shoots around the periphery of each tree. Blossom stage was rated in March using a 1–9 scale adopted from the S-97 project (5), where 1 = buds in prepink stage and 9 = postbloom stage. Fruit maturity and size were determined at the first harvest (by commercial crew) for each cultivar. Maturity was estimated using a 1–9 scale, where 1 = less than 20% and 9 = more than 90% of fruit at harvestable stage. An estimate of average size was obtained by examining harvested fruit for each tree, arbitrarily selecting several fruit that appeared average in size, and then measuring their diameter. After the final harvest for the season, the total weight of fruit harvested per tree was calculated.

Decline and nonviral diseases. Extent of bark splitting was determined in February using a 1–9 rating, where 1 = no visible splits and 9 = splits maximum in number and size on trunk and extended to scaffold limbs. Trees were examined in March for discoloration of the trunk in the cambial area due to cold injury and for bacterial canker caused by *P. s. syringae*. Cambial browning due to cold was usually uniform over wide areas of the trunk and discolored areas due to bacterial infection were restricted to elongate cankers with definite margins. Ratings of 1–9 were made as described previously (33) for trunk cambial browning (1 = healthy tissue and 9 = dead or dying tissue) and for bacterial canker (1 = no bacterial canker and 9 = entire tree killed). Trees were rated for peach tree fungal gummosis caused by *Botryosphaeria dothidea* (Moug.:Fr.) Ces. & De Not. (29,30) in December. Ratings of 1–9 were based on the frequency of gum exuded from bark infections at lenticels; thus, 1 = no visible gum spots on tree and 9 = maximum number of spots on trunk and scaffold limbs. Information concerning tree death was recorded at the end of each season and percent mortality during a 3-yr period was calculated.

Data analysis. For each characteristic or nonviral disease studied, comparisons were made between trees that tested positive for PNRSV and trees that tested negative. The number of trees testing positive or negative on each rootstock in orchards I, IIA, and IIB were considered too low for comparisons within rootstock; therefore, data were combined and subjected to analysis of covariance with rootstock as a covariable. Since orchard III consisted of only two rootstocks, analysis of variance was performed for each. Mortality data were analyzed using the chi-square test.

RESULTS

The percentages of trees in orchards I, IIA, IIB, and III that initially tested positive for PNRSV were 41.1, 26.0, 33.5, and 63.0%, respectively. The average annual increase in the percentage of PNRSV-infected trees (out of the total number of trees initially assayed) ranged from 3.6% for orchard I to 17.4% for orchard III. The overall proportions of Redhaven, Loring, and Redskin trees that tested positive initially were 57.7, 6.2, and 19.6%, respectively. The average annual increases in incidence for the same cultivars as detected by ELISA were 11.1, 4.5, and 3.8%, respectively. At least some trees in each rootstock group tested positive for PNRSV. All tests in 1986 for PDV were negative.

The number of trees assayed and the percentage of trees positive for PNRSV within orchard, cultivar, and year are presented in Table 1. Characteristics relating to growth, fruiting, and disease for trees that tested positive were compared with those that tested negative. Differences between means are indicated in Table 2, but mean values are not shown.

Mean trunk circumference of trees for a given orchard, cultivar, and year ranged from 25.7 to 55.2 cm. Although statistical differences were detected in only five of 18 cases at $P \leq 0.05$ ($0.16 \leq r \leq 0.28$) (Table 2) and in three more at $P \leq 0.1$ ($0.1 \leq r \leq 0.17$), mean values for trees tested positive for PNRSV were consistently greater (1.1–11.1%) than those that tested negative, except for Redhaven on Siberian C rootstock in orchard III. Trunk growth based on increased circumference over a 3-yr period was compared for PNRSV-infected and noninfected trees in orchards I, IIA, and IIB. For trees in orchard III, such comparisons were feasible after 2 yr, but not after 3 yr when 98.2% of the trees tested positive. According to analysis of covariance, reduction in growth associated with a positive test was shown only with Loring trees in orchard I ($P = 0.007$, $r = 0.24$). For this cultivar, the mean girth increase was 5.6 cm for PNRSV-infected trees and 8.7 cm for noninfected trees.

Differences were few and inconsistent for other characteristics related to vegetative growth (Table 2). For example, PNRSV-infected Redhaven trees in orchard IIB defoliated later in one year but earlier in the next year than the noninfected trees. Rootstock suckers for Loring in orchard I were more numerous for PNRSV-infected than for noninfected trees in two separate years (11.8 and 2.8, respectively, in 1985; 8.3 and 2.8 in 1986). The differences were highly significant ($0.0002 \leq P \leq 0.003$; $0.24 \leq r \leq 0.29$) even though not all rootstock

Table 1. Enzyme-linked immunosorbent assay detection of Prunus necrotic ringspot virus (PNRSV) in four peach orchards in three successive years

Location[a] Orchard	Cultivar[b]	Rootstock[b]	Year planted	No. trees planted	Assays for PNRSV[c] Year 1 No. trees	Year 1 Percent positive	Year 2 No. trees	Year 2 Percent positive	Year 3 No. trees	Year 3 Percent positive
USDA										
I	Redhaven	8	1976	192	158	76.6	150	81.3	113	93.8
I	Loring	8	1976	192	161	6.2	159	8.2	126	16.7
IIA	Redhaven	7	1981	112	111	34.3	111	47.7	108	55.6
IIA	Redskin	7	1981	112	112	17.9	112	24.1	112	27.7
FVSC										
IIB	Redhaven	7	1981	112	112	45.5	111	55.0	107	72.0
IIB	Redskin	7	1981	112	112	21.4	111	23.4	111	26.1
III	Redhaven	Lovell	1980	96	95	62.1	94	72.3	93	100.0
III	Redhaven	Siberian C	1980	96	91	63.7	83	73.5	76	96.1

[a] Orchards were located at the USDA Research Laboratory, Byron, GA, and Fort Valley State College (FVSC), Fort Valley, GA.
[b] Orchards I, IIA, and IIB each consisted of two different cultivars on seven or eight different peach seedling rootstocks; planting III consisted of one cultivar on two rootstocks.
[c] Orchard I was assayed in spring or early summer from 1984 to 1986; all other orchards were assayed from 1985 to 1987. Number of trees assayed is equal to number of live trees at time of assay.

types reacted by producing suckers. However, in orchard IIA, where rootstock suckers were few, Redhaven trees testing positive for PNRSV in 1985 had fewer rootstock suckers than those testing negative (0.05 and 0.59, respectively).

Flower bud density and blossom stage appeared to be little affected by PNRSV, with differences for either observed only once (Table 2). However, differences in fruit maturity were shown between infected and noninfected trees in all three cases in which ratings were made. For Redhaven in orchard IIB in 1985, fruit on the PNRSV-infected trees matured earlier, and an interaction with rootstock was indicated. In orchard III, fruit of infected Redhaven trees on Lovell rootstock ripened earlier ($P = 0.0004$; $r = 0.35$), but those on Siberian C rootstock ripened later ($P = 0.019$; $r = 0.24$) than on noninfected trees. Mean values for infected and noninfected trees on Lovell were 5.4 and 4.2, and on Siberian C, 6.4 and 7.2, respectively.

Fruit yield was significantly less for PNRSV-infected trees in three of nine cases in which yield was measured. For Loring in orchard I, fruit yield from infected trees was 47.3% lower than from noninfected trees (7.8 compared with 14.8 kg per tree; $P = 0.001$; $r = 0.35$). In orchard III, mean values for yield were consistently lower for the infected trees (8.2–18.3%), but statistical differences were found only in 1986 ($0.01 \leq P \leq 0.03$; $0.22 \leq r \leq 0.29$). Mean yields in 1986 for infected and noninfected trees on Lovell were 15.7 and 17.1 kg per tree, and for those on Siberian C, 13.8 and 16.9 kg per tree, respectively.

Trunk cambial browning was affected by PNRSV in five cases ($0.14 \leq r \leq 0.23$), but differences were inconsistent and small, never more than 1.3 points on the 1–9 point rating scale. Bark splitting was consistently greater for PNRSV-infected trees in three out of eight instances where ratings were made. Differences were highly significant for Lovell rootstock in orchard III during 1985 (mean of 3.88 compared with 2.11; $P = 0.0009$; $r = 0.33$), but in the other two cases, mean values differed by only three-tenths of a rating point.

Based on ELISA, PNRSV had little or no effect on bacterial canker (average rating for different orchards ranged from 2.0 to 6.0) or fungal gummosis (average rating ranged from 1.5 to 6.0). Bacterial canker ratings were higher in one case and gummosis ratings were higher or lower in two cases. Means of the nonviral disease ratings for PNRSV-infected and noninfected trees never differed by more than 1.1 points on the 1–9 rating scale.

In determining whether PNRSV was related to mortality, trees that initially tested negative but later positive were disregarded. Thus, the presence or absence of PNRSV in trees used was constant for the duration of the study. From 1984 to 1986, 30.8% of Redhaven trees and 23.4% of Loring trees in orchard I died. Mortality among rootstocks varied, therefore data from combined rootstocks within each cultivar were compared using the chi-square test (Table 3). Trees not infected with PNRSV tended to have a higher mortality than those infected, but no significant difference was detected at $P \leq 0.05$. In orchards IIA and IIB, death of Redhaven trees from 1985 to 1987 amounted to 10.5 and 7.1%, respectively, and death of Redskin trees was 0 and 2.8%, respectively. When data for Redhaven trees in these duplicate orchards were combined (Table 4), mortality during 3 yr was 15.7% for noninfected trees but only 2.3% for trees infected with PNRSV. This was significant according to the chi-square test ($P = 0.02$). In orchard III, which consisted of all Redhaven trees on two different rootstocks, 2.5% of trees on Lovell rootstock died in 1985 or 1986, whereas 18.3% of trees on Siberian C rootstock died during this period. For Siberian C, 11 out of 59 PNRSV-infected trees (or 18.6%) died, and four out of 23 noninfected trees (or 17.4%) died. No statistical difference was detected between these groups.

DISCUSSION

ELISA is strain-specific and may not have detected all strains of PNRSV present. However, the antiserum used was prepared against PNRSV-G, which virtually has the status of type strain for this virus. ELISA results were partly verified by performing bioassays for selected trees in each orchard using Shirofugen cherry (*Prunus serrulata* Lindl.). Sera against PNRSV-G have been used by a number of other workers (1,10,16,27,28), giving indications of activity to a broad range of strains of PNRSV.

Based on tree circumference, there appeared to be a slight trend toward decreased growth of PNRSV-infected trees. The time of infection is unknown for all trees that were positive in the initial assays. Infection may have been due to bud or seed transmission at the nursery or it may have occurred after

Table 2. Yearly comparison of growth, fruiting, and disease for trees tested for Prunus necrotic ringspot virus (PNRSV) by enzyme-linked immunosorbent assay in four peach orchards[a]

	Orchard I[b]						Orchard IIA[b]				Orchard IIB[b]				Orchard III[c]			
	1984		1985		1986		1985		1986		1985		1986		1985		1986	
Characteristics	RH	L	RH	L	RH	L	RH	RS	RH	RS	RH	RS	RH	RS	L	S	L	S
Vegetative growth																		
Trunk circumference, cm	<	–	<<	–	–	<	–	–	–	–	–	–	–	–	<	–	<	–
Vegetative bud rating	–	–	–	–	···	···	–	–	···	···	>*	–	···	···	–	–	···	···
Defoliation rating	–	–	–	–	–	*	–	*	–	–	<	–	>	–	–	–	–	–
Rootstock suckers, no.	–	>>**	–	>>**	–	–	<<	**	–	>	–	–	**	–	–	–	–	–
Fruit production																		
Flower bud density, no./10 cm	···	···	···	···	···	···	–	–	···	···	–	>>*	···	···	–	–	···	···
Blossom stage rating	–	–	–	–	···	···	–	–	···	···	–	–	···	···	–	>	···	···
Fruit maturity rating	···	···	···	···	···	···	···	···	···	···	>*	···	···	···	>>	<	···	···
Fruit size, cm diam.	···	···	···	···	···	···	···	···	···	···	–	···	···	···	–	–	···	···
Fruit yield, kg/tree	–	<<	···	···	···	···	–	···	···	···	–	···	**	···	–	–	<	<
Decline and diseases																		
Cambial browning rating	–	**	<	–	*	>	–	*	–	–	>>	<	*	–	>	–	–	–
Bark cracking rating	···	···	···	···	···	···	–	>**	···	···	–	–	>	–	>>	–	···	···
Bacterial canker rating	–	–	–	–	–	–	–	–	–	–	–	–	**	–	–	–	>	–
Gummosis rating	–	–	–	<	*	–	*	**	–	–	–	–	–	–	>	–	–	–

[a] Based on analysis of covariance (orchards I, IIA, and IIB) or analysis of variance (orchard III), < and << indicate that the mean for PNRSV-infected trees is lower than that of the noninfected trees at $P \leq 0.05$ and $P \leq 0.01$, respectively; likewise, > and >> indicate that the mean for infected trees is higher than that of noninfected trees at these significance levels. * and ** indicate an interaction between virus and rootstock at $P \leq 0.05$ and $P \leq 0.01$, respectively. A minus sign indicates data were collected and analyzed, but no difference or interaction was detected. Approximate numbers of trees that were compared can be determined from Table 1.

[b] Orchards I, IIA, and IIB consist of two cultivars (RH = Redhaven, L = Loring, RS = Redskin) on several rootstocks, which are combined.

[c] Orchard III consists of one scion (RH) on two rootstocks (L = Lovell, S = Siberian C) that are presented separately.

planting. Redhaven trees, which showed the highest incidences of PNRSV, either had higher incidences at the time of planting or were more susceptible to virus spread than the other cultivars. During 3 yr of study that began when trees were 5- to 9-yr old, a negative effect on growth was indicated only with the cultivar Loring in orchard I. Results might have been different had trees been studied at a younger age or for a longer duration. Pine (23) showed that when 3-yr-old peach trees were graft-inoculated with PNRSV and measured for 4 yr, trunk growth was reduced by 12–51% depending on the virus source. In Japan (34), some peach and nectarine cultivars that were propagated with PNRSV-infected budwood had a slightly reduced trunk growth over the first 3 yr in nursery plots and others were unaffected. Perhaps growth is affected by PNRSV to a much greater extent during the early acute stage of infection (19) than during the longer chronic phase when the virus is latent. In our study, most of the trees evaluated may have been in the latent phase.

In a survey conducted in central Georgia by Wells (32), tree decline symptoms frequently associated with the presence of PNRSV included basal sprouts and bark splitting. We found that infected Loring trees had more numerous rootstock suckers, grew slower, and yielded less fruit than noninfected trees. Although not determined for Loring, bark splitting in some instances was greater for infected than for noninfected trees.

Yamaguchi et al (34) reported that maturity of fruit on trees infected with PNRSV tended to be 2–3 days earlier than for noninfected trees. We found that fruit maturity was earlier or later depending on rootstock. The trend toward reduced fruit yields due to PNRSV agrees with studies in other peach-producing areas (6,25,26,34).

We did not find a consistent relationship between PNRSV and factors associated with PTSL, such as trunk cambial browning or bacterial canker. Whereas Redhaven tended to be more susceptible to death caused by PTSL than the other cultivars (5), it also had the highest incidences of PNRSV according to ELISA, as was the case in a survey of several peach cultivars in South Carolina (27). However, these observations apparently are independent, and no evidence was found to implicate PNRSV as a contributing factor in tree death. In fact, PNRSV might have prolonged the life of Redhaven trees in orchards IIA and IIB, but data are insufficient to conclude this. Considering that some viruses induce resistance (12), a mild or latent strain of PNRSV might affect resistance to factors or agents that shorten tree life. Helton and Hubert (9) demonstrated that PNRSV induces systemic resistance in prune trees to invasion by *Cytospora cincta*.

Our study includes characteristics and decline problems of peach trees that have not been reported previously in relation to infection by PNRSV. The overall effects of the virus, based on ELISA detection with antiserum prepared against PNRSV-G, were minimal and accounted for only a small portion of the variation observed. However, reductions in fruit yields due to PNRSV were substantial in some cases. It must be remembered that the previous history of PNRSV infection in each orchard is unknown. Differences in the time of initial infection of trees could have resulted in variation that would affect these comparisons. Considering that interactions between virus and rootstock were frequently detected, use of virus-free propagation material in rootstock evaluation programs is important.

Table 3. Mortality in peach orchard I during 3 yr (1984–1986) as related to enzyme-linked immunosorbent assay (ELISA) detection of Prunus necrotic ringspot virus (PNRSV)[a]

		Redhaven		Loring	
Rootstock	ELISA[b]	No. dead/ total	Percent dead	No. dead/ total	Percent dead
Halford	+	7/23	30.4	0/0	···
Halford	−	0/0	···	4/22	18.2
Harrow	+	0/21	0	0/0	···
Harrow	−	1/2	50.0	2/19	10.5
Lovell	+	2/16	12.5	0/4	0
Lovell	−	2/3	66.7	0/14	0
NA-8	+	5/10	50.0	0/1	0
NA-8	−	6/10	60.0	10/18	55.6
Nemaguard	+	7/15	46.7	0/1	0
Nemaguard	−	2/3	66.7	1/22	0
Siberian C	+	3/5	60.0	0/0	···
Siberian C	−	0/0	···	4/5	80.0
NC NRL-4	+	3/13	23.1	0/1	0
NC NRL-4	−	0/4	0	7/19	36.8
NC 152-AI-2	+	6/17	35.3	1/3	33.3
NC 152-AI-2	−	0/1	0	6/20	30.0
Combined[c]	+	33/120	27.5	1/10	10.0
Combined	−	11/23	47.8	34/139	24.5

[a]Trees that tested negative in 1984 but were later positive were omitted.
[b]Symbols + and − indicate positive and negative tests, respectively, for PNRSV.
[c]Data for combined rootstocks for PNRSV-infected and noninfected trees were compared using a chi-square test; no differences were detected at $P \leq 0.05$.

Table 4. Mortality of Redhaven peach trees in orchards IIA and IIB during 3 yr (1985–1987) as related to enzyme-linked immunosorbent assay (ELISA) detection of Prunus necrotic ringspot virus (PNRSV)[a]

		Mortality by orchard (no. dead/total)		Mortality for combined orchards	
Rootstock	ELISA[b]	IIA	IIB	No. dead/ total	Percent dead
Halford	+	0/4	1/3	1/7	14.3
Halford	−	1/5	2/5	3/10	30.0
Lovell VF	+	0/0	0/7	0/7	0.0
Lovell VF	−	1/14	0/6	1/20	5.0
Nemaguard	+	0/5	0/12	0/17	0.0
Nemaguard	−	1/8	0/1	1/9	11.1
Tennessee RL	+	0/8	0/15	0/23	0.0
Tennessee RL	−	1/5	0/0	1/5	20.0
S-60	+	0/4	0/2	0/6	0.0
S-60	−	4/8	1/9	5/17	29.4
NC 503I-4	+	0/9	1/5	1/14	7.1
NC 503I-4	−	0/4	0/8	0/12	0.0
NC 88I-2AC-2	+	0/7	0/7	0/14	0.0
NC 88I-2AC-2	−	1/5	1/5	2/10	20.0
Combined[c]	+	0/37	2/51	2/88	2.3
Combined	−	9/49	4/34	13/83	15.7*

[a]Trees that tested negative in 1985 but were later positive were omitted.
[b]Symbols + and − indicate positive and negative tests, respectively, for PNRSV.
[c]Data for combined rootstocks for PNRSV-infected and noninfected trees were compared using a chi-square test; asterisk indicates difference at $P \leq 0.05$.

LITERATURE CITED

1. Barbara, D. J., Clark, M. F., Thresh, J. M., and Casper, R. 1978. Rapid detection and serotyping of Prunus necrotic ringspot virus in perennial crops by enzyme-linked immunosorbent assay. Ann. Appl. Biol. 90:395-399.
2. Cameron, H. R. 1977. Effects of viruses on deciduous fruit trees. HortScience 12:484-487.
3. Clark, M. F., and Adams, A. N. 1977. Characteristics of the microplate method of

enzyme-linked assay for the detection of plant viruses. J. Gen. Virol. 34:475-483.
4. Cole, A., Mink, G. I., and Regev, S. 1982. Location of Prunus necrotic ringspot virus on pollen grains from infected almond and cherry trees. Phytopathology 72:1542-1545.
5. Dozier, W. A., Jr., Knowles, J. W., Carlton, C. C., Rom, R. C., Arrington, E. H., Wehunt, E. J., Yadava, U. L., Doud, S. L., Ritchie, D. F., Clayton, C. N., Zehr, E. I., Gambrell, C. E., Brittain, J. A., and Lockwood, D. W. 1984. Survival, growth, and yield of peach trees as affected by rootstocks. HortScience 19:26-30.
6. Heaton, C. R., Ogawa, J. M., and Nyland, G. 1981. Evaluating economic losses caused by pathogens of fruit and nut crops. Plant Dis. 65:886-888.
7. Helton, A. W. 1974. Effects of Prunus ringspot virus on growth and productivity of Richards Early Italian prune trees. Phytopathology 64:1179-1181.
8. Helton, A. W. 1975. Effect of cultivar and rootstock on incidence of viral and nonviral symptoms in PRSV-infected *Prunus domestica*. Phytopathology 65:539-541.
9. Helton, A, W., and Hubert, J. J. 1968. Inducing systemic resistance to *Cytospora* invasion in *Prunus domestica* with localized Prunus ringspot virus infections. Phytopathology 58:1423-1424.
10. Jaffee, B. A., Powell, C. A., and Derr, M. A. 1986. Incidence of Prunus necrotic ringspot virus in some Pennsylvania peach orchards and nurseries. Plant Dis. 70:688-689.
11. Klos, E. J., and Parker, K. G. 1960. Yields of sour cherry affected by ringspot and yellows viruses. Phytopathology 50:412-415.
12. Kuć, J. 1983. Induced systemic resistance in plants to diseases caused by fungi and bacteria. Pages 191-221 in: The Dynamics of Host Defense. J. A. Bailey and B. J. Deverall, eds. Academic Press, New York.
13. Kyotani, H., Yoshida, M., and Yamaguchi, M. 1983. Infection of Prunus necrotic ringspot virus (PRSV) in stone fruits. I. Influence of PRSV on peach culture. Pages 17-26 in: Bull. Fruit Tree Res. Stn. Ser. A (Yatabe), no. 10.
14. Milbrath, J. A. 1950. Latent ringspot virus of cherries reduces growth of nursery trees. Plant Dis. Rep. 34:374-375.
15. Milbrath, J. A. 1957. Effect of some sour cherry viruses on growth of young orchard trees. Phytopathology 47:655-657.
16. Mink, G. I. 1980. Identification of rugose mosaic-diseased cherry trees by enzyme-linked immunosorbent assay. Plant Dis. 64:691-694.
17. Mink, G. I., and Aichele, M. D. 1984. Use of enzyme-linked immunosorbent assay results in efforts to control orchard spread of cherry rugose mosaic disease in Washington. Plant Dis. 68:207-210.
18. Mink, G. I., and Aichele, M. D. 1984. Detection of Prunus necrotic ringspot and prune dwarf viruses in Prunus seed and seedlings by enzyme-linked immunosorbent assay. Plant Dis. 68:378-381.
19. Nemeth, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff, The Hague, Netherlands. 840 pp.
20. Nyland, G., Gilmer, R. M., and Moore, J. D. 1974. Prunus ringspot group. Pages 104-132 in: Virus Diseases and Noninfectious Disorders of Stone Fruits in North America. U.S. Dep. Agric. Handb. 437. U.S. Government Printing Office, Washington, DC.
21. Parker, K. G., Brase, K. D., Schmid, G., Barksdale, T. H., and Allen, W. R. 1959. Influence of ringspot virus on growth and yield of sour cherry. Plant Dis. Rep. 43:380-384.
22. Parker, K. G., and Cochran, L. C. 1951. Similarities of symptoms produced by the viruses causing ringspot of peach and necrotic ringspot of sour and sweet cherry. Phytopathology 41:942.
23. Pine, T. S. 1964. Influence of necrotic ringspot virus on growth and yield of peach trees. Phytopathology 54:604-605.
24. Ritchie, D. F., and Clayton, C. N. 1981. Peach tree short life: A complex of interacting factors. Plant Dis. 65:462-469.
25. Saunier, R. 1972. Incidences d'une virose du type ringspot sur la comportment de deux cultivars du pecher. Pomol. Fr. 14:175-185.
26. Schmitt, R. A., Williams, H., and Nyland, G. 1977. Virus diseases can decrease peach yields. Cling Peach Q. 13(1):17-19.
27. Scott, S. W. 1989. Incidence of Prunus necrotic ringspot virus in selected peach orchards of South Carolina. Plant Dis. 73:913-916.
28. Torrance, I., and Dolby, C. A. 1984. Sampling conditions for reliable routine detection by enzyme-linked immunosorbent assay of three ilarviruses in fruit trees. Ann. Appl. Biol. 104:267-276.
29. Weaver, D. J. 1974. A gummosis disease of peach trees caused by *Botryosphaeria dothidea*. Phytopathology 64:1429-1432.
30. Weaver, D. J. 1979. Role of conidia of *Botryosphaeria dothidea* in the natural spread of peach tree gummosis. Phytopathology 69:330-334.
31. Weaver, D. J., Wehunt, E. J., and Dowler, W. M. 1974. Association of tree site, *Pseudomonas syringae*, *Criconemoides xenoplax*, and pruning date with short life of peach trees in Georgia. Plant Dis. Rep. 58:76-79.
32. Wells, J. M., Kirkpatrick, H. C., and Parish, C. L. 1986. Symptomatology and incidence of Prunus necrotic ringspot virus in peach orchards in Georgia. Plant Dis. 70:444-447.
33. Yadava, U. L., Doud, S. L., and Weaver, D. J. 1984. Rating scales to assess cold injury and bacterial canker development in peach trees in the field. HortScience 19:645-648.
34. Yamaguchi, M., Yoshida, M., and Kyotani, H. 1983. Infection of Prunus necrotic ringspot virus (PRSV) in stone fruits. II. Influence of PRSV on peach culture. Pages 27-37 in: Bull. Fruit Tree Res. Stn. Ser. A (Yatabe), no. 10.