

Influence of Recent and Chronic Virus Infections on Strawberry Growth and Yield

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ABSTRACT

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The spread of viruses into strawberry (*Fragaria × ananassa* 'Hood'), and the effects of these viruses on growth and yield were measured under field conditions in the Willamette Valley, Oregon, over a 37-mo period. Strawberry mottle, mild yellow-edge, and crinkle viruses were the most common of those detected and usually occurred together. The percentage of infected plants increased yearly, reaching 91% in the 3rd yr. Recent virus infections (those that occurred

within 1 yr prior to the sampling date) did not significantly reduce the number or weight of fruit per plant compared to that of healthy plants in 1973 and 1974. In contrast, during the same period, fruit number, weight of fruit per plant, and weight of plant tops after harvest were significantly reduced (16, 19, and 17%, respectively) by chronic virus infections (those occurring more than 1 yr before sampling) when compared to recently infected plus healthy plants.

The North American literature published since 1954 on the influence of viruses on strawberry growth and yield was reviewed in 1970 (5). Recent studies on this topic in the Pacific Northwest have utilized known virus cultures as inoculum for field tests (1, 8) and for greenhouse tests (10), or have used natural field sources of inoculum for greenhouse yield tests (12). Strawberry cultivars of the Pacific Northwest also have been ranked for field tolerance to virus infection (4).

We have initiated a series of investigations of the effects of viruses on the cultivated strawberry. In the present study, we report effects of recent and chronic virus infection on growth and yield of Hood strawberry under field conditions in the Willamette Valley of Oregon. Plants were allowed to become naturally infected in the field so that the influence of different strains known to occur in many strawberry viruses (5) could be exerted in the experiment.

MATERIALS AND METHODS

Certified Hood strawberry plants from Washington were planted at the North Willamette Experiment Station, Aurora, Oregon, on 3 June 1971. Eighteen plants from the lot used for planting were indexed by petiole-insert leaf grafting to *Fragaria vesca* var. *semperflorens* (Dcne.) Ser. 'Alpine', from which other leaves had been removed just before grafting (7). The plants were found to be free of viruses detectable in this indicator plant (6). Twelve test plots, totaling 120 plants, were selected

within the experimental planting. Each plot consisted of two adjacent rows of five plants each. Loss of plant samples caused the totals to vary from year to year during the study. Plants were spaced 38 cm apart, in rows 107 cm apart. Four buffer rows were maintained between plots, and buffer plants were grown at the ends of the plots. Standard commercial management practices were used in the planting, except that aphicidal sprays were discontinued after 1972. Plants were grown in the single-hill system, and runners were kept removed except for those used for indexing. Fruit yields from individual plants and number of fruit per plant were obtained in 1972, 1973, and 1974. Yield records taken in 1972 were incomplete. In July 1974, the test plants were dug and the top growth of each plant was severed from the crown at a point immediately above the topmost root and was individually weighed.

Sample runner plants (generally two per mother plant) were taken from each of the 120 Hood plants in November 1971. Sample runner plants about 4 wk old also were taken from all previously healthy plants in July of 1972, 1973, and 1974. The runner plants were grown in a screened greenhouse in which systemic aphicides were routinely used. They were indexed by leaf grafting on Alpine strawberry. In evaluation of runner plants (9), mother plants that had become infected each year by graft-transmissible viruses were identified. Strawberry crinkle virus (CV), strawberry mottle virus (MV), and strawberry mild yellow-edge virus (MYEV) were detected most often and usually occurred together. We shall assume that all of the diseases involving the graft-transmissible agents detected in this study had viral etiology.

RESULTS

Over the 37 mo of the experiment, a combination of CV, MV, and MYEV was found in 62% of infected plants. Double- and single infections of these viruses accounted for 20% more. Strawberry necrotic shock, a disease reportedly associated with tobacco streak virus (13), was present in combination with CV, MV, or MYEV in 11% of the infected plants. Vein banding virus, either alone or in combination with CV, MV, or MYEV made up the remaining 7%.

Because CV, MV, and MYEV occurred together in most of the infected plants, data were pooled from all infected plants when assessing the effects of viruses on growth and yield, differentiating among virus-infected plants only according to the length of time they had been infected.

In November 1971, 5 mo after planting, 6% of the plants were virus infected (Table 1). During the next 8 mo an additional 36% became infected. Only 9% of the plants indexed virus-free after 37 mo in the field.

The 13 plants sampled in 1972 for yield (Table 2) either were infected when previously indexed (chronic infections), or were infected since they had last been indexed (recent infections). There were no statistically significant yield differences between the two groups of plants.

Yields for 1973 were separated into three classes according to virus status and duration of infection of the

plants: (i) healthy when indexed in 1973; (ii) recent infections, infected since July 1972; and (iii) chronic infections, infected when previously indexed. There were significant differences in number of fruit per plant and total yield of fruit per plant between chronically infected and healthy plants (Table 2).

Yields for 1974 were separated into classes similar to those for 1973 yields. There were marked differences between chronically infected and healthy plants, and these differences were paralleled by differences in fresh weight of tops of plants harvested in July 1974 (Table 2).

Differences were not always significant ($P=0.05$) when several classes were used. Therefore, the data for 1973 and for 1974 were each pooled into two classes for further statistical comparisons using the *t*-test: (i) chronic infections, as defined above; and (ii) healthy, plus recent infections as defined above. In 1973, the number of fruit per plant in chronically infected plants was significantly reduced (by 18%) below the number of fruit produced in healthy plus recently infected plants (Table 3). The same pattern occurred in 1973 for weight of fruit per plant (19% reduction). In 1974, the pattern was repeated with weight of fruit per plant (19% reduction) and weight of plant tops after harvest (17% reduction), but the reduction in number of fruit per plant (15%) was significant only at $P=0.10$.

In comparing overall effects of chronic infection versus lack of infection in 1973 and 1974, the percentage reduction in number of fruit per plant was 19%; in total yield, 21%; and in reduction of fresh weight of plant tops, 23%.

DISCUSSION

Our observations confirm an earlier report (3) that strawberry virus infection in Oregon is not uniform from year to year in the same location. The 6% infection during the summer and fall of 1971 was probably introduced by alate aphids coming into the young planting. The 36% infection for 1972 and the 20% infection in 1974 represent major seasonal variations in the amount of virus infection. The reasons for these variations are not understood. Variation in levels of alate aphid vectors of strawberry viruses moving within and into the test field, as well as variations in local apterous aphid populations, may be factors. Populations of strawberry aphids are known to vary considerably from year to year in the Pacific Northwest (11).

Barritt and Loo (1), working in Puyallup, Washington,

TABLE 1. Virus infection of Hood strawberry, Aurora, Oregon, 1971-1974

Date ^a runners taken from field	No. of plants			Cumulative infection (%)
	Recently virus- infected ^b	Chronically virus- infected	Healthy	
Nov 1971	7	0	113	6
Jul 1972	39	7	65	41
Jul 1973	39	46	34	71
Jul 1974	18	85	10	91

^aTwo runner plants from each mother plant in the field were transplanted to a screenhouse and subsequently indexed for virus content as described in the text. Loss of runner plants during indexing accounts for the variation in yearly totals.

^bAll virus-infected plants were grouped together to obtain these counts, regardless of the identity of the viruses.

TABLE 2. Yield and growth of Hood strawberry plants indexing free from viruses, recently virus-infected, or chronically virus-infected at time of harvest, Aurora, Oregon, 1972-1974

Virus status	1972			1973			1974			Weight of plant top in July (g)
	Plants sampled (no.)	Fruit (no.)	Plant weight (g)	Plants sampled (no.)	Fruit (no.)	Plant weight (g)	Plants sampled (no.)	Fruit (no.)	Plant weight (g)	
Healthy	-	-	-	34	51	554	10	97	640	491
Recent infections ^a	7	21	290	39	49	514	19	92	628	437
Chronic infections ^b	6	23	245	46	<u>41</u> ^c	<u>430</u>	84	80	518	377

^aVirus infected for less than 1 yr when yield records were taken.

^bVirus infected for more than 1 yr when yield records were taken.

^cUnderline indicates the value was significantly less ($P=0.05$) than the value for healthy in the same column.

TABLE 3. Comparison of harvest records and plant weight of chronically virus-infected plants versus healthy, plus recently infected, Hood strawberry plants, Aurora, Oregon, 1973-1974

Virus status	1973		1974		Weight of plant top (July) (g)
	Fruit per plant		Fruit per plant		
	(no.)	wt (g)	(no.)	wt (g)	
Chronic ^a infections	41	430	80	518	377
Healthy+recent ^b infections	50	533	94	632	455
Significance of <i>t</i> -test ^c	*	*	N.S.	*	*

^aVirus infected for more than 1 yr at the time yield records were taken.

^bVirus infected for less than 1 yr at the time yield records were taken.

^cAt $P = 0.05$, * = significant; N.S. = not significant.

with Hood strawberry chronically infected with virulent virus strains, found that MV or MYEV alone or together had little effect on yield. Strawberry crinkle virus alone caused a significant yield reduction (30%); all three viruses together reduced yield by 60%. The differences in magnitude of yield loss in Hood at Puyallup and at Aurora, Oregon, may have arisen from differences in environment and in virus strains. Strawberry plants at Puyallup became 100% infected in 3.5 mo (1), compared with 91% infection at Aurora after 37 mo. These two studies indicate a much more rapid rate of virus infection in the Puyallup Valley than in the central Willamette Valley.

Mottle, vein banding, and latent C viruses caused progressive yield decline in several strawberry cultivars in a recent study in eastern Canada (2). In the present study, there was a definite difference in yield loss in Hood strawberry between plants recently infected and chronically infected mainly by CV, MV, and MYEV. More detailed studies are needed to determine the length of time required for viruses to cause their maximum effects on strawberry growth and yield. Nevertheless, if the reaction of Hood strawberry is typical, the consequences of chronic virus infection must be distinguished from those of recent virus infections in future studies of the effects of viruses on growth and yield in strawberries. Seasonal variation in the amount of virus infection and delays in the onset of severe effects of viruses on fruit yield might be used to advantage in designing management schemes to optimize economic returns in commercial strawberry plantings.

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