# Competitive Relationships in Virus-Infested Sugar Beet Fields

R. S. Loomis and C. W. Bennett[1]

*Received for publication March 17, 1966*

In a community of plants of high population density, each individual is severely restricted in growth by competition with its neighbors for required environmental factors. Nutrients, water and other edaphic components commonly are most limiting in natural environments, but in agricultural fields, where such deficiencies may be eliminated easily, fluxes of carbon dioxide and radiant energy for photosynthesis frequently become the major limiting factors (3, 4, 8).[2]

Individual plants in a community differ in their physiological or morphological characteristics due to differences in genetic composition, or to chance and manipulated local variations in population density. Such differences permit some individuals to compete for limiting factors more efficiently and thus make better growth and contribute relatively more to the ultimate yield of the field. These superior individuals may tend to compensate for the poorer growth of their more suppressed neighbors and thus maintain yield near the same level that would be attained with uniform plants.

Variations in competitive ability also may occur because some plants in a field are diseased. De Wit (3) describes work by Reestman which revealed a situation where healthy potato plants were able to compensate for the poorer growth of competing plants diseased with leaf roll. In this instance, Watson and Wilson (13), among others, consider that leaf roll reduces leaf area and photosynthesis so that the compensation was probably due to reduced competition for light. In a disease situation, the possible occurrence and degree of compensation obviously is a complex function of many things, including disease reaction, stage of plant growth when the attack occurs, basic morphology of the parts affected, plant density and limiting environmental factors. While the altered patterns of competition may be difficult to unravel, the overall effects may have economic and biologic significance. Of particular interest with crop plants is whether economic yields may be maintained at high levels up

[1] Associate Professor of Agronomy, University of California, Davis and Plant Pathologist, Emeritus, U. S. Department of Agriculture, Salinas, California.

[2] Numbers in parentheses refer to literature cited.

to some critical proportion of diseased plants. Disease infestations up to this critical frequency could be tolerated economically.

The research reported here was a study of competition in sugar beet fields infested with the beet yellows virus (BYV). Data relating overall root yield to level of infestation by BYV can be obtained from naturally infested commercial fields (5). However, there are very real experimental difficulties in sampling portions of fields, or groups of fields, and ascertaining which part of the variability in yield is due to some regression on the frequency of diseased plants. This is especially true in California where time of infection may vary, and where several strains of this and other viruses differing in virulence may occur within the same field (2). A simpler approach may be used in which varying proportions of plants in a disease-free field are hand-inoculated with a highly virulent strain of BYV. An inherent assumption is that populations infested naturally, with possibly less virulent strains, would react similarly but to a lesser degree.

M. A. Watson and her associates (11, 12) carried out an extensive series of experiments at Rothamsted with BYV, including some studies with varying proportions of hand-inoculated plants. They concluded from examination of total root yields that compensation did not occur. As De Wit (3) points out, total yields provide little information on the nature of competitive relationships if both members in the competition react, e.g., if one is suppressed while the other is released. We attempted to circumvent this problem by measuring the component yields of both healthy and diseased plants.

## Methods and Materials

Two experiments were conducted in the field at Davis, California, during 1962 and 1963, with sugar beets. The crops were planted in late May after the spring peak of the local flights of the BYV vector, the green peach aphid vector [*Myzus persicae* (Sulz.)] (6). Late planting permitted the experiment to be conducted with vigorous, disease-free plants. The variety 'Spreckels 202H' which shows no specific resistance to the virus was used. The crops were planted on 14-26-in double-row beds (20 in average row spacing) for furrow irrigation and thinned by hand to single plants on 8 in centers. The experiments were conducted at this single density of about 35,000 plants per acre.

In each year, a randomized complete block design (with 4 replicates in 1962 and 5 replicates in 1963) with seven inoculation treatments was used. The treatments consisted of inoculating

in late June, 0, 17, 33, 50, 67, 83, or 100% of the plants in a uniform pattern in the various plots. Inoculation was accomplished in the early morning by transferring to each plant to be infected, a leaf piece carrying about 10 green peach aphids. The leaf pieces came from source plants grown in a greenhouse at Salinas and carried strain 5 of the beet yellows virus (2). It is our experience that while the aphids feed long enough to transmit the virus, they fail to migrate to adjacent plants or even to survive under the high summer temperatures at Davis. However, to insure that the inoculations were confined to the selected plants, 'Metasystox' aphicide was applied to the entire field on the day following inoculation. Strong symptoms (vein clearing and stunting of younger leaves, yellowing of old leaves) were apparent on the inoculated plants within one month post-inoculation.

The validity of the treatments was maintained fairly well in 1962 when the final harvest was made at an early date (Table 1). There may have been a tendency to overestimate the number of diseased plants when their frequency was low, but generally the observation of greater numbers of diseased plants than intended inoculations appeared due to some natural infections by visiting aphids and to over-inoculation.

In 1963, there was more variation between the number of inoculations and the frequency of diseased plants estimated at harvest. Some alate green peach aphids were noted on the crop in early June and 'Metasystox' was applied topically to reduce natural infections. When inoculations were made on June 27,

Table 1.—Details of the two competition experiments.

| Year | Planting date | Inoculation | | Final harvest date | Nitrogen applied lbs/acre | No. of replication |
|---|---|---|---|---|---|---|
| | | Date | Stage | | | |
| 1962 | May 9 | June 27 | 12 to 16 leaves | Sept. 13 | 180 | 4 |
| 1963 | May 23 | June 27 | 8 to 10 leaves | Oct. 22 | 200 | 5 |

| 1962 | Treatment | | | | | | |
|---|---|---|---|---|---|---|---|
| | A | B | C | D | E | F | G |
| Percent of plants inoculated: | 0 | 17 | 33 | 50 | 67 | 83 | 100 |
| Percent of diseased plants as observed at final harvest: | 3±1 | 28±2 | 42±6 | 61±7 | 65±10 | 80±3 | 97±1 |

| 1963 | Classification group | | | | | |
|---|---|---|---|---|---|---|
| | I | II | III | IV | V | VI |
| Percent of diseased plants as observed at final harvest: | 6±2 | 19±3 | 40±2 | 52±2 | 69±2 | 89±3 |

five plants among 200 counted had symptoms of mosaic virus and three had symptoms of yellow viruses. Thus, the background level of natural infections was higher than in 1962. The hand inoculations were less effective (none of the completely inoculated plots were classified as having more than 94% diseased plants at final harvest, Table 1). This was due, in part, to small, late-emerging plants which escaped inoculation. The field was rethinned several times but a number of these plants remained and had to be accounted for at final harvest. Difficulty also was encountered due to the later harvest and a marginal deficiency of nitrogen that occurred in some plots late in the season. The experiment had been carried longer into the fall period in order to accentuate growth differences. The yellowing symptoms, related to senescence and low nitrogen, interfered with proper classification of the plants. These difficulties led to variations in the frequency of diseased plants measured within each treatment and led to a consideration of the 1963 data on the basis of an unreplicated, completely random design. All plots were ranked on the basis of observed frequencies. Three plots, having extreme values for yield in relation to disease frequency, were eliminated. The remaining plots were grouped by frequency rank in natural classes having no more than a 13% range in frequency. These groups are shown near the bottom of Table 1.

A key tactic of the experiments, conducted on a fertile, well drained soil, was to maintain water and nitrogen (commonly limiting factors in the Davis environment) at high levels through irrigation and supplemental fertilization (180 to 200 lb N/A). Petiole analysis (10) revealed that nitrogen and phosphorus were not limiting during the growing season and there was no basis for suspecting deficiencies of other elements. However, in 1963, some plots showed low nitrogen near harvest time. Since Watson and Watson (12) concluded that photosynthesis (leaf area and net assimilation rates) was affected by the virus, we attempted by these tactics to create a situation where competition for light would be most intense.

Each plot was two beds (four rows wide and 50 feet long). At harvest, an experienced person sorted the plants harvested from 30 feet of the center two rows, into diseased and non-diseased groups based on visual symptoms. Data were collected on the two classes separately.

Tare, sucrose and dry matter determinations were made on two subsamples of storage roots from each plot by the Spreckels Sugar Company. Yields of fresh and dry tops also were determined. The "tops", as in the commercial sense, included a small

portion by weight of stem and adhering dead leaves. Leaf areas were estimated in 1963 from the dry weights of leaf punches taken equally from leaf blades harvested from six plants in each plot. A 60°-angle wire frame was placed over each of these plants, with the apex of the frame at the apical meristem; leaves whose petioles fell within this sector were harvested for determination of leaf area.

## Results

Growth curves for the extremes in treatments (0 and 100% inoculated) from the two experiments are shown in Figure 1. In both instances, a highly significant depression in root growth was evident within 5 weeks after inoculation. Total root yield, and the difference between healthy and diseased plants, continued to increase for the duration of the experiments. The ultimate reduction in root yield was 28% for 1962 and 33% for 1963. The yields of fresh tops increased to a maximum in September in both years; there were no significant differences between the A and G treatments at any time in either season.

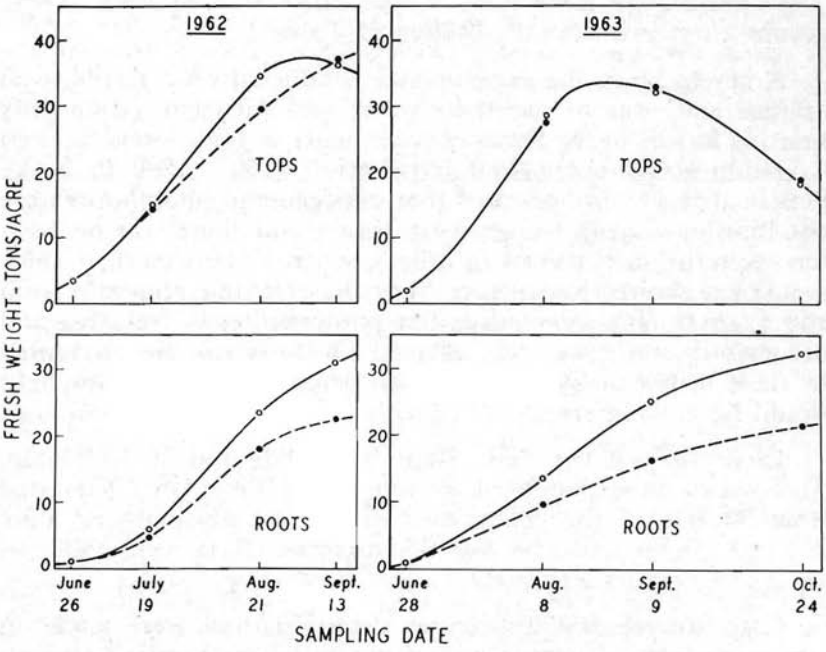


**Figure 1.—Growth of storage roots and tops of sugar beet crops at Davis, California in 1962 and 1963. (Solid line, 0% inoculated with beet yellows virus; dashed line, 100% inoculated. Means of 3 to 7 observations.)**

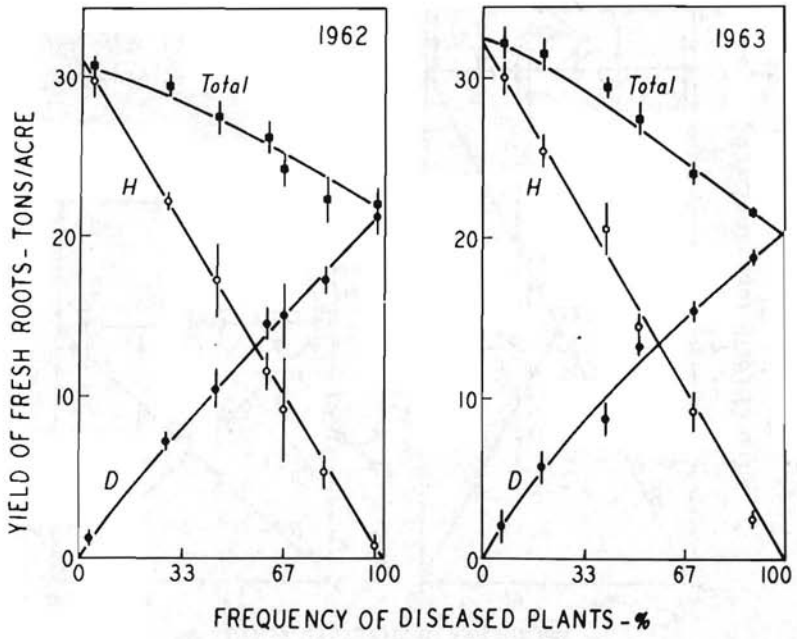


**Figure 2.—Total yield of storage roots (T) and the component yields of healthy (H) and diseased (D) of sugar beet plants at various disease frequencies for the two experiments.**

In both years, similar curves were obtained by regressions through individual plot values or through means of treatments or rank groups. In Figure 2, the mean root yields observed at the final harvest are plotted against the corresponding mean frequency of diseased plants. In these and subsequent figures, eye-fitted curves are shown. Regressions were calculated in several cases which seemed to have curvilinear responses. Generally the quadratic component was not significant. For example, in Figure 2, linear regressions provide a significant fit for all lines except for the yield of roots from diseased plants in 1963, where the quadratic term was barely significant. The tendency shown for compensation in total yield at low levels of diseased plants was also evident in 1962.

Total yield of fresh tops and the component yields of diseased and healthy plants were not affected by disease (Figure 3). Both groups contributed equally to the total yield, which was constant for all levels of disease. Data on yields of dry tops (not shown) were similar. However, despite the equal weight, the diseased

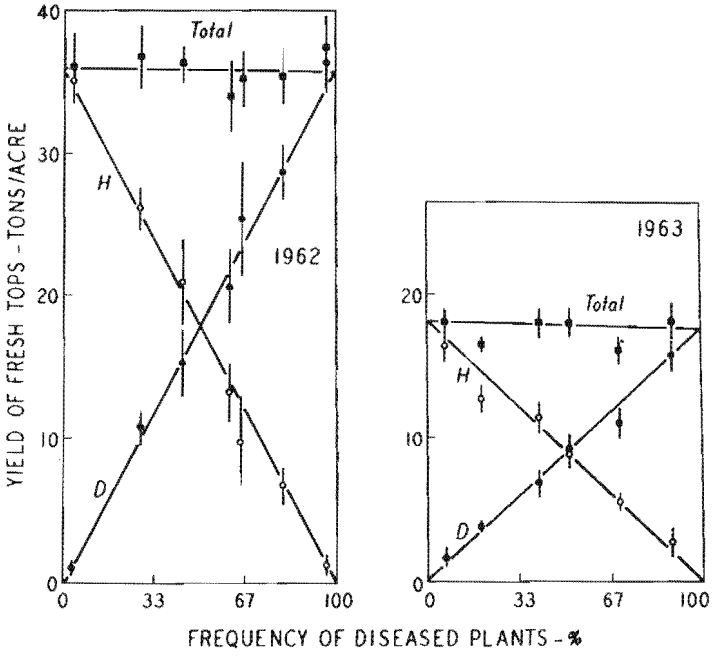


**Figure 3.—Total and component yields of fresh tops of sugar beet plants.**

plants had fewer and smaller leaves than the competing healthy plants (Table 2). Thus, the leaf area of diseased plants was less (Figure 4). The reduction in leaf area per plant was least for plants competing with other diseased plants; the difference increased as the proportion of healthy plants in the competition increased. The infected plants were relatively free of infections by other pathogens such as *Cercospora* leaf spot and did not

**Table 2.—Number of living leaves on healthy and diseased sugar beet plants at final harvest, their mean area and specific leaf weights. 1963 data.**

| | | Mean percent of diseased plants in group | | | | | |
|---|---|---|---|---|---|---|---|
| | | 6 | 19 | 40 | 52 | 69 | 89 |
| No. of livings leaves per plant | Healthy: | 44 | 46 | 52 | 49 | 46 | — |
| | Diseased: | | 45 | 43 | 46 | 42 | 45 |
| Mean area per leaf in $cm^2$ | Healthy: | 180 | 140 | 180 | 170 | 160 | |
| | Diseased: | | 120 | 110 | 120 | 120 | 140 |
| $cm^2\ g^{-1}$ Specific leaf weight dry weight | Healthy: | 190 | 200 | 180 | 160 | 180 | |
| | Diseased: | — | 180 | 180 | 190 | 170 | 150 |

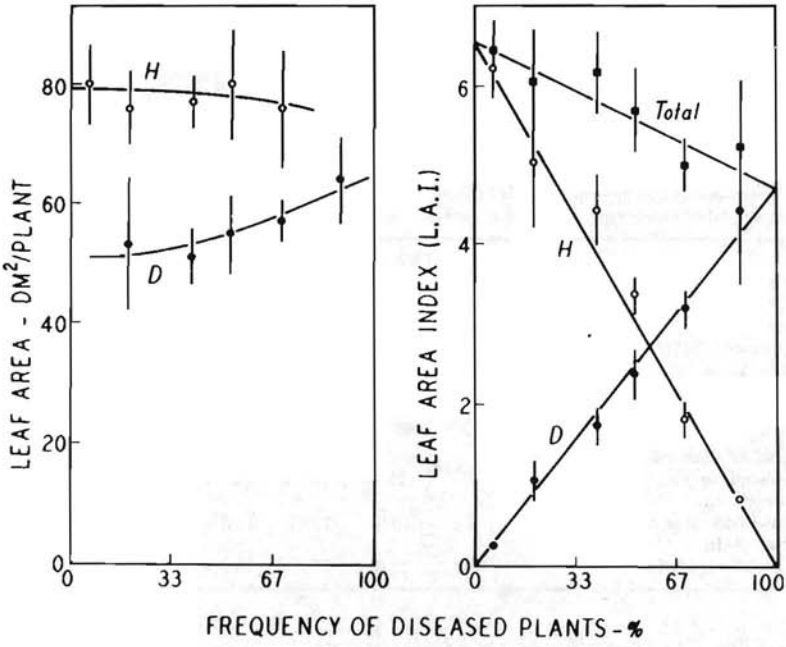


**Figure 4.—Left. Leaf area per plant for healthy and diseased sugar beet plants at various frequencies of disease, 1963 data. Right. Total and component leaf area indices, 1963 data.**

experience any noticeable stress for water or nutrients or by extremes of temperature; such factors have been observed to accentuate the loss of leaf area from plants infected with BYV.

The leaf area index (LAI: ratio of leaf area to ground area) as extrapolated in Figure 4, ranged from 4.7 with 100% diseased plants to 6.5 with 100% healthy plants. Here again, only the linear components of the regressions for total and component leaf area indices were significant.

Sucrose yields are presented in Figure 5 and dry matter totals (roots and tops combined as an estimate of total biological yield) are shown in Figure 6. In neither instance was there any significant deviation from linearity despite some tendencies towards curvilinearity. Sucrose yields reflect the yields of fresh roots quite closely, since sucrose concentration (Table 3) was unaffected by the disease. The decline in sucrose yields with increasing frequency of diseased plants was relatively greater than was the decline in total dry matter since the yield of tops was not reduced by disease. Thus the coefficient of economic yield

was reduced by the disease in direct proportion to the frequency of diseased plants. In 1963, 41% of the harvested dry matter of healthy plants occurred as sucrose as compared to 31% in the diseased plants (extrapolated values).

**Table 3.—Mean sucrose concentrations, fresh basis, in sugar beet roots for healthy and diseased plants competing in varying ratios.**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | 1962 | | | | | |
| Percent of diseased plants in the treatment: | 3 | 28 | 42 | 61 | 65 | 80 | 97 |
| Sucrose % in diseased plants: | — | 11.7 | 12.1 | 12.0 | 12.0 | 12.2 | 11.8 |
| Sucrose % in healthy plants: | 12.5 | 11.9 | 11.8 | 12.2 | 11.8 | 11.8 | — |
| | | 1963 | | | | | |
| Percent of diseased plants in the group: | 6 | 19 | 40 | 52 | 69 | 89 | |
| Sucrose % in diseased plants: | — | 13.6 | 13.4 | 13.5 | 13.5 | 13.2 | |
| Sucrose % in healthy plants: | 13.2 | 13.6 | 13.6 | 13.6 | 13.4 | — | |

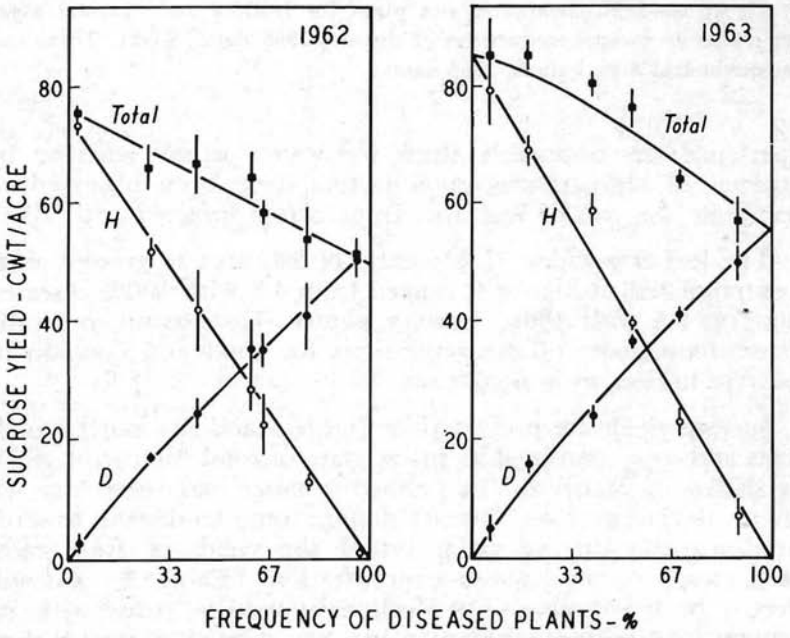


**Figure 5.—Total and component yields of sucrose in storage roots of sugar beets.**

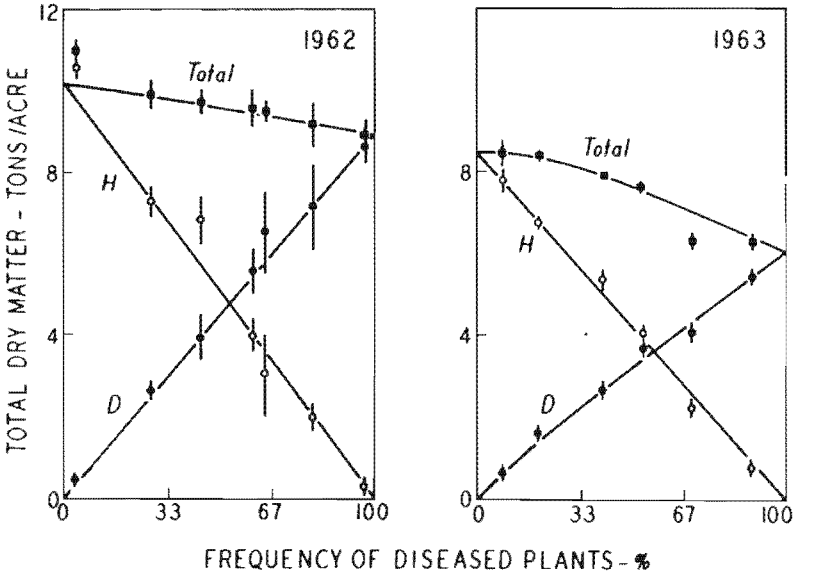


Figure 6.—Total and component yields of dry matter of sugar beet storage roots and tops combined.

## Discussion

A tendency towards compensation by the healthy plants for the poorer growth of the adjacent diseased plants was observed in both experiments, particularly in 1963. The interpretation of this tendency (significant only in the case of root yields from diseased plants) offers certain difficulties since the compensation apparently was made by the diseased plants. Putting aside for the moment the possibility of experimental bias as a cause for the result, then a "Montgomery effect" occurred (3, 4). Stated simply, the diseased plants did better in competition with healthy plants than in competition with other diseased plants. The data which were collected offer no basis for explaining how a Montgomery effect might take place.

There was no evidence of competition in the soil environment, and the diseased plants had lower leaf areas displayed in the aerial environment. It appears that the generally lower growth of the diseased plants was related to their less efficient competition in the aerial environment. The smaller leaf area and low photosynthesis rates in diseased leaves (12) may be sufficient to account for the effects observed.

A Montgomery effect cannot be explained by assuming that the leaf area of the healthy plants was too high, perhaps exceeding some optimum (4), since the heavily diseased stands which had lower leaf area, also had lower growth. Furthermore, an optimum has not been observed in crop growth–LAI curves for sugar beet up to LAI 8 to 12 in other environments (4; Worker and Loomis, unpublished). The diseased plants may have had some advantage in leaf display. The limited notes, measurements and photographs which were made of the foliage canopies revealed only that in mixed competition, the diseased leaves seemed to be displayed similarly to the non-diseased leaves. Leaf area was not limiting for light interception, since even on October 10, all of the canopies absorbed at least 97% of the irradiance at solar noon.

It seems more likely that the slight compensatory effect was an artifact resulting from misclassification of healthy and diseased plants. The misidentification in each plot of two or three large, healthy plants with many yellow, old leaves would account for the differences noted. For the present, we must conclude that the experimental methods were not adequate to determine small differences in competition. However, it is clear that marked compensation did not occur. In general, competition was equal and the healthy and diseased plants contributed to total yield as an approximately linear function of their relative frequencies. Thus the yield reductions noted with BYV are closely related to the number of diseased plants in the population.

Donald (4) recently has emphasized that interplant competition may affect internal "competition" for available nutrients and substrates. Thus, interplant competition for light due to variations in light interception, which in turn affects the amount of carbohydrate available to roots, will alter the patterns of plant development. With the beet yellows virus, root growth was reduced markedly. The question arises whether this was due to some direct effect of the virus on root growth or to an indirect effect of "intraplant competition" for the substrates necessary for root growth.

Storage root growth is regulated in part by the amount of carbohydrate translocated from the shoot; when water, inorganic nutrients, or other factors are not limiting, then sucrose concentration in the roots remains static and root growth varies with carbohydrate supply. If root growth is restricted directly, as by plant water deficit or nitrogen deficiency, then sucrose accumulates and its concentration in the root increases (7). In the present experiments, sucrose concentration in the roots was

not affected by the virus, or by competition between healthy and diseased plants, although mean root yields were reduced 30% by the disease. Thus, it may be concluded that the virus did not limit root growth either directly or by restricting the supply of some substrate other than sucrose or by altering the supply of hormones (e.g., auxin) which regulate root growth.

This conclusion differs from that reached by other workers (1, 9, 11), who generally have reported that the virus caused a decrease in sucrose concentration. A hypothesis accounting for this discrepancy can be developed from an understanding of nitrogen deficiency responses. With a smaller supply of nitrogen than was available to the crops in these experiments, a period of nitrogen deficiency would have occurred near the end of the season, causing a reduction in root growth and an increase in sucrose concentration. Since healthy and diseased plants differ in total growth, and hence in nitrogen absorption and assimilation, it would be reasonable to expect that nitrogen depletion would develop more slowly (and later) with diseased plants than with healthy plants. In 1963, the mean concentration of $NO_3$-N in petioles concentration in the fully inoculated plots was 2400 ppm (dry basis) on October 14, 1963 in contrast to a concentration of 1000 ppm in uninoculated control plots. Thus, under low nitrogen conditions, the presence of virus would not be expected to reduce sucrose concentration but rather to prevent it from increasing as rapidly as in healthy plants. This interpretation fits with the relatively high sucrose concentration noted in other experiments (1, 9) since such sucrose levels generally are not attained except when nitrogen is deficient. Watson *et al.* (11) did not observe a strong nitrogen × disease interaction, perhaps because both levels of nitrogen which they used allowed the sugar beet plants to become severely deficient by the end of the season, thus permitting maximum sucrose concentrations to be reached in diseased as well as in healthy plants.

In many field environments, interactions might be expected between the BYV and other factors. Thus, stress by high temperature, or for water and nitrogen and additional infection by *Cercospora* leaf spot have been observed to accentuate the loss in leaf area which occurs with BYV.

These competition studies were conducted at a single high plant population and with only one virulent strain of the virus. These conditions were chosen as likely for the demonstration of compensation. However, in many places infection with virus

may occur in the seedling stages and viral strains more virulent than strain 5 have now been identified. Thus, while compensation and changes in sucrose concentration were not observed in a single system with well nourished plants free of secondary problems, they might occur as general phenomena under other conditions.

## Summary

Hand inoculation with a virulent strain of the beet yellows virus was made to a disease-free sugar beet crop. Losses in yields of storage roots and sucrose were found to be proportional to the frequency of diseased plants. There was a slight tendency for yield compensation only at low frequencies of disease. This apparently was due to better growth by the diseased plants in competition with healthy plants than by those in competition with diseased plants. However, the experimental approach was not sufficiently sensitive to establish whether this was a real effect. The principal conclusion of the experiments is that no appreciable compensation occurred. Sucrose concentrations in beet roots were unaffected by the virus; arguments are presented that any major effects on root either in yield or in sucrose percentage, are indirect.

## Acknowledgment

We wish to acknowledge the expert assistance of Mrs. Jean Washington, who made the separations of healthy and diseased plants and assisted in summarizing the data. This study was supported in part by a grant from the beet sugar industry of California.

### Literature Cited

(1) BENNETT, C. W., C. PRICE and J. S. MCFARLANE. 1957. Effects of virus yellows on sugar beet with a consideration of some of the factors involved in changes produced by the disease. J. Am. Soc. Sugar Beet Technol. 9 (6): 479-494.

(2) BENNETT, C. W. 1960. Sugar beet yellows disease in the United States. U. S. Department of Agriculture Tech. Bull. 1218. 63 p.

(3) DE WIT, C. T. 1960. On competition. Versl. Landbouwk. 66 (8): 1-82.

(4) DONALD, C. M. 1963. Competition among crop and pasture plants. Adv. Agron. 15: 1-118.

(5) DUFFUS, J. E. 1963. Incidence of beet viruses in relation to overwintering beet fields. Plant Disease Rept. 47: 428-431.

(6) HILLS, F. J., W. H. LANGE, R. S. LOOMIS, H. L. HALL and J. L. REED. 1965. Sprays for aphid control increase sugar beet yields in Davis tests. Calif. Agr. 19 (3): 6-7.

(7) LOOMIS, R. S. and D. J. NEVINS. 1963. Interrupted nitrogen nutrition effects on growth, sucrose accumulation and foliar development of the sugar beet plant. J. Am. Soc. Sugar Beet Technol. 12 (4): 309-322.

(8) LOOMIS, R. S. and W. A. WILLIAMS. 1963. Maximum crop productivity: An estimate. Crop Sci. 3: 67-72.

(9) MCFARLANE, J. S., C. W. BENNETT and A. S. COSTA. 1954. Effect of virus yellows on the yield and sucrose percentage of the sugar beet at Salinas, California. Proc. Am. Soc. Sugar Beet Technol. 8 (1): 215-218.

(10) ULRICH, A., D. RIRIE, F. J. HILLS, A. G. GEORGE and M. D. MORSE. 1959. Plant analysis: A guide to sugar beet fertilization. Univ. Calif. Agr. Exp. Sta. Bull. 766. 24 p.

(11) WATSON, M. A., D. J. WATSON and R. M. HULL. 1946. Factors affecting the loss of yield of sugar beet caused by beet yellows virus. I. Rate and date of infection; date of sowing and harvesting. J. Agr. Sci. 36: 151-166.

(12) WATSON, D. J. and M. A. WATSON. 1953. Comparative physiological studies on the growth of field crops. III. The effect of infection with beet yellows and beet mosaic viruses on the growth and yield of the sugar-beet root crop. Ann. Appl. Biol. 40: 1-37.

(13) WATSON, D. J. and J. H. WILSON. 1956. An analysis of the effects of infection with leaf-roll virus on the growth and yield of potato plants, and of its interactions with nutrient supply and shading. Ann. Appl. Biol. 44: 390-409.

(14) WATSON, D. J. 1958. The dependence of net assimilation rate on leaf area index. Ann. Bot. N.S. 22: 37-55.