Reaction of Resistant and Susceptible Sugar Beet Cultivars to Beet Yellows and Beet Western Yellows Viruses

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ABSTRACT

Sugar beet hybrids, 'US H9B' (resistant) and 'US H7A' (susceptible), and an open-pollinated breeding line, 'C13' selected for resistance to beet yellows and beet western yellows viruses and used as a pollen parent for US H9B, were tested for resistance to virus transmission by inoculation with few to many apterous green peach aphids. Increasing numbers of apterae increased virus infection to a similar degree with the three cultivars, hence resistance to aphid transmisssion was not demonstrated.

Cultivar C13 exhibited internal plant resistance to both

viruses as it showed relatively less reduction in root yield with increasing levels of disease than did the other cultivars. US H9B produced superior root yields at all levels of infection but relatively little of its superior performance appeared to be due to virus resistance. The selection procedure for the development of C13 apparently also resulted in the selection of heritable characteristics other than yellows resistance which contribute to the superior performance of its hybrids.

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Additional key words: virus transmission, aphid transmission, crop losses, resistance.

The sugar beet (Beta vulgaris L.) hybrid 'US H9B' has been bred for resistance to beet yellows virus (BYV) and beet western yellows virus (BWYV) and has been outstanding in sugar yield when infected lightly or severely by these viruses (2, 4). In a 1968 test at Davis, California, this hybrid, with natural virus infection, gave a greater root yield than the similar but nonresistant hybrid, 'US H7A'. US H9B was colonized by fewer apterous green peach aphids, Myzus persicae (Sulzer), suggesting that resistance might be due to resistance to virus transmission (1). Present experiments were designed to test this hypothesis. A second objective was to assess the contribution of resistance to yellows to the yield of this cultivar.

MATERIALS AND METHODS.—US H9B and US H7A are three-way hybrids. Both have the same F_1 hybrid (C562 CMS \times 546) as a male sterile parent but different pollen parents (3). The pollen parent of US H9B is 'C13', a fifth generation selection for resistance to BYV and BWYV (5).

Field trials were conducted at the Broom's Barn Experimental Station, Suffolk, England and at Davis, California in 1970, and at Davis in 1971.

The strain of BYV used at Broom's Barn was propagated in sugar beet. It occurs throughout beet growing areas of England and produces pronounced vein clearing in sugar beet a week or so after inoculation.

The 'Colusa' strain of BYV, which induces severe symptoms on sugar beet and *Chenopodium capitatum* (L.) Asch., was used at Davis in 1970. Strains of BYV and BWYV used in the 1971 experiment at Davis were those used by McFarlane et al. (4) in the

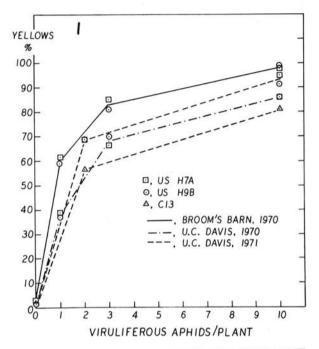
development of breeding line C13. BYV was propagated in New Zealand spinach (*Tetragonia expansa* Murr.) and BWYV in "White Icicle" radish (*Raphanus sativus* L.).

The *M. persicae* used for virus inoculations at Davis were of a biotype selected by W. H. Lange, Department of Entomology, University of California, Davis, for efficient transmission of beet yellows virus. Aphids were propagated on chinese cabbage [Brassica pekinensis (Lour.)] and transferred to virus infected plants 24-48 hours before being used for inoculation. Viruliferous aphids were placed on individual plants with a small brush at Davis. At Broom's Barn, aphids were allowed to crawl onto plants from small test tubes into which the aphids had been transferred 1-2 hr previously.

Plots at Broom's Barn were five rows (2.54 m) X 5.94 m and at Davis four rows (3.05 m) X 9.14 m. At both locations plants were spaced 25 to 30 cm within a row. The 1970 treatments were a factorial combination of four aphid numbers per plant (0, 1, 3, and 10 aphids) X two cultivars (US H9B and US H7A) with six replications at Broom's Barn and four at Davis. Main plots were aphid levels per plant and subplots were cultivars. Treatments at Davis in 1971 were all combinations of three aphid numbers per plant (0, 2, and 10) X two viruses (BYV and BWYV) X three cultivars (US H9B, US H7A, and C13). The design was a split-split plot with four replications. Main plots were aphid levels in randomized complete blocks, subplots viruses, and sub-subplots were cultivars.

Disease was evaluated by observation of about 70 plants within each plot. The same plants were

harvested for yield and quality determinations. Symptomless plants were tested as possible carriers of BYV by feeding non-viruliferous apterae (M. persicae) on leaf sections in large plastic petri dishes for one day at room temp. Ten to 20 of these aphids were caged on each of three seedlings of C. capitatum.



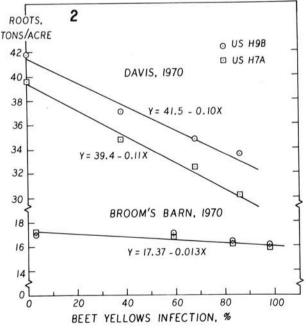


Fig. 1-2. 1) Effect of numbers of apterous Myzus persicae on infection of sugar beet cultivars by beet yellows virus. 2) Effect on root yield of increasing percent infection by beet yellows virus. Counts of infected plants were made 8 weeks after inoculation.

RESULTS.—Virus transmission.—Plants inoculated with BYV at Broom's Barn showed vein clearing symptoms as early as 10 days following inoculation and were severely yellowed 4 weeks after inoculation. There were no obvious differences in symptoms on the two hybrids. At Davis, typical yellowing symptoms were obvious by the fourth week following inoculation and a few plants inoculated with BYV had cleared veins. When inoculated with beet yellows by ten aphids per plant, C13 was darker green in color than the two hybrids and in some plots US H9B was noticeably greener than US H7A. BYV symptoms were slight on C13 and often difficult to detect. Symptoms of BWYV appeared slowly and on relatively few plants.

Non-inoculated plots at Davis remained essentially virus free throughout the season, because, as usual, aphids died out in hot weather. In England, where aphid activity increases during the summer, half of the plots were sprayed with insecticide on 11 June, 1-2 days following inoculation. On 15 June aphids were counted on 10 plants in each plot. There were few aphids on sprayed or non-sprayed plants and no indication that one cultivar favored their multiplication more than another. All plots were sprayed with insecticide on 10 July. However, subsequent aphid activity resulted in 4, 17, and 70% infection in non-inoculated plots, respectively, on 13 July, 3 August, and 10 September.

Eight weeks after inoculation, zero, one, three, or ten apterae per plant gave progressively greater percentages of plants with yellows and infected US H7A and US H9B with equal efficiency at Broom's Barn and Davis in 1970 (Fig. 1). At Davis in 1971, zero, two, and ten aphids per plant, gave similar results but cultivar C13 showed 12% fewer infected plants than the hybrids when inoculated with BYV by two or ten aphids per plant.

If a cultivar is resistant either to aphid transmission or virus infection, inoculating it with few aphids per plant should result in a lower percent infection than with a susceptible cultivar, but this difference should decrease when more aphids are used for inoculation. In these tests none of the cultivars was resistant to infection by BYV but some plants of C13 remained symptomless although infected by BYV. Bioassays from symptomless plants inoculated by 10 aphids per plant in the 1971 tests resulted in the following recoveries of BYV: US H7A, 56% (five out of nine plants assayed); US H9B, 67% (10 out of 15); and C13, 74% (14 out of 19). Recovery of BYV from a greater percentage of symptomless plants of C13 and US H9B, the vague symptoms, and the darker green coloring of infected plants of these cultivars indicate that the smaller counts of diseased plants reported from field trials may be due to symptom masking rather than resistance to aphid vectors or to virus infection.

Symptoms of BWYV may be suppressed by high temperatures and high nitrogen fertility (7) such as occurred in the 1971 Davis experiment. Ten weeks after inoculation only 24% of the plants of US H7A exposed to ten aphids per plant showed symptoms

TABLE 1. Effect of inoculation with beet yellows virus and beet western yellows virus by two and ten green peach apterae/plant on the root yield of sugar beet cultivars, University of California, Davis, 1971

	Root yield (fresh), tons/acre							
		Virus and no. viruliferous aphids/plant						
	No virus	BYV			BWYV			
Cultivar		2	10	$\overline{\mathbf{x}}^{\mathbf{a}}$	2	10	$\bar{\mathbf{x}}^{\mathbf{a}}$	
C13	32.0	27.1	26.2	26.7	32.2	31.7	32.0	
US H7A	33.6	25.8	23.6	24.7	32.2	30.1	31.2	
US H9B	36.3	27.8	27.6	27.7	35.2	33.6	34.4	

compared with US H9B 12% and C13 2%. Plants of US H7A, US H9B, and C13 with two aphids had, respectively, 10, 4, and 2% with symptoms. Symptomless plants were not tested for BWYV but by analogy with BYV, the near absence of C13 plants with symptoms is more likely to be due to symptom masking than to plants remaining free from virus infection due to resistance to infection.

Root yield.—At Broom's Barn both hybrids yielded significantly less with an increasing proportion of plants infected by BYV but US H9B did not yield more than US H7A as it did at Davis in both years (Fig. 2 and Table 1) and in other field trials (2, 4). The failure of US H9B to yield better may be attributed to a greater susceptibility to powdery mildew which was severe from late August on. On 10 September about 10% of the plants of US H7A were covered by powdery mildew in contrast to nearly 100% of US H9B.

The smaller apparent effect of BYV on root yield at Broom's Barn compared to Davis was undoubtedly the result of the natural spread of virus to noninoculated control plants as the season progressed.

At Davis in 1970, BYV inoculation with zero, one, three, and ten aphids per plant resulted in levels of infection which were closely correlated with root yield. The cultivar X disease-level interaction for root

yield was not significant, indicating that US H9B yielded as much more than US H7A at all levels of BYV infection (Fig. 2). Statistical tests of the slopes of the regression lines of Fig. 2 show that they are not significantly different which also indicates that increasing levels of BYV infection affected the two hybrids similarly.

In the 1971 experiment which included the cultivar C13 and BWYV, there was a statistically significant interaction between cultivars and numbers of aphids per plant for both viruses (Tables 1 and 2). With either virus, however, the only significant component of the interaction was a significantly larger difference between either of the hybrids and C13 when healthy than when diseased. For example (Table 1), healthy US H9B - C13 = 36.3 - 32.0 = 4.3tons/acre; but when diseased, US H9B - C13 = 27.6 -26.7 = 1.0 ton/acre. The latter is a significantly smaller difference which demonstrates more resistance in C13 than in US H9B. Similar comparisons between US H9B and US H7A with either virus result in differences that are not significantly different. For example, healthy US H9B - US H7A = 36.3 - 33.6 = 2.7 tons/acre and with BYV, 27.6 - 24.7 = 3.0 tons/acre. This lack of significant difference in response to yellows supports the conclusion that US H9B is as superior to US H7A when healthy or diseased. Thus, this experiment clearly demonstrates resistance in C13 to both viruses but not in US H9B.

Percent sucrose.—BWYV had no significant effect on the concentration of sucrose in beet roots. In both trials at Davis, BYV infection significantly decreased the sucrose content of roots. The cultivars did not have significantly different percent sucrose and all were affected similarly by BYV. Both trials gave a significant linear regression of percent sucrose on percent of plants with BYV and each indicated a 0.1 percentage point decrease in percent sucrose for each 10% increase in plants with BYV.

DISCUSSION.—A lack of resistance to virus infection by apterous green peach aphids does not preclude the possibility that C13 and US H9B may possess a leaf constituent which disturbs aphid

TABLE 2. Mean squares for orthogonal components of cultivars × diseaselevel interaction for sugar beet root yield, University of California, Davis, 1971

		Mean so	quares ^a
Source of variation	df	BYV	BWYV
Cultivar ^b × aphid no./plant ^c	4	13.5245**d	9.0117*d
$(C13 \text{ vs H7} + \text{H9}) \times (0 \text{ vs A2} + \text{A10})$	1	45.3270**	29.4700**
$(H7 \text{ vs } H9) \times (0 \text{ vs } A2 + A10)$	1	4.5601	3.9161
$(C13 \text{ vs H7} + H9) \times (A2 \text{ vs A10})$	1	0.1102	2.3852
$(H7 \text{ vs } H9) \times (A2 \text{ vs } A10)$	1	4.1006	0.2756

^aError mean square (36 df) = 3.0577. Coefficient of variation = 5.7%.

 $^{^{}b}H7 = US H7A, H9 = US H9B.$

c0 = not inoculated, A2 and A10 = inoculation by two or ten aphids/plant.

d*, ** = significant at the 5 and 1% levels, respectively.

feeding and which could result in less efficient transmission by alatae, the principal carriers of BYV and BWYV to sugar beet crops. The existence of such a leaf constituent has been postulated by W. H. Lange (University of California, Davis, California, personal communication) and may result in plants escaping

infection by alatae.

Since half of the genome of US H9B is from C13 and since C13 clearly demonstrates internal resistance to both BYV and BWYB, one would expect the hybrid to have some resistance, but this is not clearly supported by these experiments. The regressions of Fig. 2 and mean yields for disease versus nondiseased plants of Table 1 furnish estimates as to the percentage of improved yield of US H9B over US H7A that might be attributed to disease resistance. Percent improved root yield due to disease resistance = {[diseased: US H9B - US H7A] - (healthy: US H9B - US H7A)]/(diseased: US H9B - US H7A)} 100. Root yields predicted from the regression equations of Fig. 2 at 0 and 80% BYV infection indicate that about 28% of the increased root yield of US H9B over US H7A might be due to resistance to BYV, [33.5 - 30.6) - (41.5 - 39.4)]/(33.5 - 30.6)100 = 28%. A second estimate is obtained from the 1971 Davis experiment, Table 1: {[(36.3 - 33.6) -(27.7 - 24.7)]/(27.7 - 24.7)} 100 = 10%. Similarly, percentage yield improvement due to resistance to BWYV is estimated from Table 1 as about 16%.

This evidence suggests that US H9B owes most of its superior performance to characteristics other than disease resistance inherited from its C13 pollen parent. Yield performance probably can be improved

considerably by the incorporation of disease resistance in the inbred components of the male sterile parent. US H9B has been tested as a triploid hybrid by doubling the chromosome number of C13 before using it as the pollen parent of US H9B (6). The triploid yielded little more roots than the diploid, indicating that US H9B performs better than US H7A due to the interaction of the genome from C13 with the genome of C562 CMS X 546 rather than to the strictly quantitative effect of the C13 genome.

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