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THE BARLEY YELLOW-DWARF VIRUS IN WHEAT: IMPORTANCE, SOURCES OF RESISTANCE, AND HERITABILITY

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SUMMARY

The barley yellow-dwarf virus (BYDV) is transmitted by several aphid species to barley, oats, wheat, triticale, and rye. The virus has world-wide distribution. Severe yield losses can be incurred, and virus is often present at low and visually unnoticeable levels in wheat. Resistant or highly tolerant varieties provide the only means of control of BYDV. The search for sources of resistance has been only moderately successful in wheat. Two sources, CI 13232 and Anza, were found to have genetic resistance to BYDV at Davis, California. Resistance is inherited quantitatively, and it is suggested that recurrent selection will be the most effective breeding method for BYDV resistance.

BRUEHL (1961) suspected that the barley yellow-dwarf virus disease (BYDV) would become established as the most widely distributed virus disease in the Gramineae. Following its discovery in California in 1951 (OSWALD and HOUSTON, 1953b), it has been observed throughout North America, Europe, Australia, and New Zealand. It seems likely that, as the symptoms of the disease become more widely known, it will be identified in other parts of the world as well. BYDV is a circulative aphid-transmitted virus. Several aphid species are known vectors. Vector-specific strains of the virus have been demonstrated (ROCHOW, 1961). The virus is somewhat host specific, but strains infecting cereals have been recovered from many grass species (OSWALD and HOUSTON, 1953a). The virus infects the various small grains - barley, oats, wheat, triticale, and rye. Barley and oats generally show greater yield losses than wheat. Symptom expression is only rarely seen on rye, whereas there is a wide range of susceptibility among triticale varieties. Average yield losses in wheat are variable and have been estimated at about 5 to 10% annually (DOODSON and SAUNDERS, 1970) in England. In 1959 BYDV was damaging to cereals throughout many areas in the U.S. (MURPHY, 1959).

Symptoms are not so pronounced in wheat as in barley or oats. Leaves of plants infected in the seedling or later stages show yellowing or reddening. Dwarfing is evident, mainly after seedling infection.

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The number and fertility of headed tillers is reduced in infected plants. This is very pronounced in space-planted conditions where the first tillers are killed and late-developing tillers give the plant an open-tillered appearance. BRUEHL (1961) and ROCHOW (1961) have described and illustrated in detail the various symptom expressions of BYDV.

Control of BYDV is relegated primarily to the development of resistant varieties. Aphid control is not effective in eliminating infection because the momentary feeding which is necessary to kill aphids with a systemic insecticide results in transmission of the virus. Insecticidal control of aphids has been effective in reducing the amount of damage in some instances by preventing secondary spread of the virus from initial (primary) foci of infection in areas where viruliferous aphids immigrate to cereal fields (CLOSE, 1970). Yield losses can be averted by control of time of planting. Early fall planting or late spring planting should be avoided in most areas because of high probability of aphid infestation on seedling plants. GILL (1970) noted that yield losses of wheat due to BYDV in Canada are often lower than losses observed in barley, which he ascribed to earlier planting for wheat. Fall infection of winter wheat caused greater yield reductions (81%) than spring infections (62%) in controlled inoculation studies in South Dakota (FITZGERALD and STONER, 1967). Early infection causes much greater yield reduction than late infection (BRUEHL, 1961; DOODSON and SAUNDERS, 1970; GILL, 1967; OSWALD and HOUSTON, 1953b; SMITH, 1967).

Breeding for resistance has been very successful in barley (SCHALLER *et al.*, 1970), where a single gene for resistance found in Ethiopian barleys was transferred by backcrossing to adapted varieties. Moderately good sources of resistance have been identified in oats, and progress is being made in developing resistant varieties.

SOURCES OF RESISTANCE

Bruehl and Damsteegt (BRUEHL, 1961) observed more than 3,000 varieties in Washington in 1960 and noted several varieties with some tolerance (Sun, Red Russian (CI4509), CI11230, Ci11234, CI11236, PI108980, and PI108981). DOODSON and SAUNDERS (1970) studied the effects of time of inoculation on four wheat varieties and found Cappelle Desprez to be most tolerant of those tested. OSWALD and HOUSTON (1953b) reported Sonora 37 to be the most tolerant California variety. Some New Zealand varieties, notably 705,01, have quite high tolerance to BYDV (SMITH, 1967).

A search for resistance was initiated at Davis, California, in 1961. More than 5,000 entries from the U. S. Department of Agriculture World Collection were evaluated by means of the screening method that had been used for barley (SCHALLER *et al.*, 1963), which utilizes spring planting (March) and visual scores of symptom expression. Aphid infestation occurs naturally, predominantly with _Rhopalosiphum padi_ (L.) and _R. maidis_ (L.), in "aphid traps", which are strips of a mixture of susceptible oat and barley varieties. The aphid traps are planted monthly beginning in September. When the test plants are in the 2- to 3-leaf stage the aphid traps having high infestation of aphids are cut, and the straw is scattered over the test plants. This method is simple and generally results in a high degree of symptom expression, so that visual ratings are quite useful. About 25 entries that had quite high tolerance were identified in the initial screening. Coker 55-9 (CI 13232 = Chancellor^2 x T. Hybrid) was one of the most promising varieties in this group.

In 1969, observations on BYDV were made in the International Spring Wheat Yield Nursery, and one entry, (Lerma Rojo x Norin 10-Brevor) x Andes$^{3}_{e}$, showed essentially no symptom expression. Because of extremely good grain yields throughout California in 1964, 1965 and 1970-72, this variety was released for production (named Anza, same as or similar to Mexicani in Sudan, Karamu in New Zealand, and WW15 in Australia; QUALSET *et al*., 1973).

Since resistance in barley was found only in collections from Ethiopia, it seemed worthwhile (SMITH, 1967) to examine Ethiopian tetraploid and hexaploid wheats. The first group of 252 tetraploids was examined at Davis in 1971 (with G. W. Bruehl). About 10 entries were relatively symptom-free and were considered worthy of further testing. Results in 1972 confirmed that some of these entries have tolerance, and additional tests are in progress. A second group of 332 entries was studied in 1972, and of these only 26 were considered worthy of further testing. At present it does not appear that Ethiopian wheats have the same degree of resistance as Ethiopian barley.

The search for resistance in wheat has not been very successful. All of the entries found up to now show some symptoms and at best must be considered tolerant rather than highly resistant. Some varieties show considerable yellowing but only little yield reduction (e.g., 705,01; SMITH, 1967). Differences in times of maturity and stages of development at the time of inoculation also cause difficulties in evaluating resistance (FITZGERALD and STONER, 1967). Several authors have noted that evaluating yield reduction due to infection by BYDV is the only reliable method of detecting tolerance. SMITH (1967) suggested that resistance be evaluated after inoculating at two stages of growth. Different test plants of the same genotype must be used for each growth stage because of a protective effect of early infection against subsequent inoculation. Because there is an apparent virus dosage effect, a larger number of aphids per plant should be used for inoculation at the later stage than at the early stage. It seems apparent now that the very high levels of resistance found in barley will not be found in wheat. The screening method used at Davis, with severe infection of seedling plants, may be too restrictive, and varieties with worthwhile tolerance may be discarded because of poor appearance. However, genotypes with good growth and limited symptom expression identifed by this method are believed to have good tolerance.

HERITABILITY OF RESISTANCE

Confounding effects of growth habit, time of infection, and variable environmental conditions reduce confidence that selection for resistant entries from the world collection has been effective. It is necessary to conduct genetic tests to determine if there is a heritable component to the variation in symptom expression. F_3 lines from crosses of CI 13232 with the early- and late-maturing susceptible varieties Ramona 50 and Big Club 60 were infected with BYDV and observed symptom development. About 8% of 800 lines from each cross were classified as resistant or highly tolerant. We have selected plants from the most promising lines and have isolated several lines in the F_8 that apparently have quite good tolerance. We have therefore concluded that CI 13232 has genetically controlled, but not simply inherited, tolerance to BYDV.

The possible genetic control of resistance in Anza has also been under investigation. Individual, space-planted, unreplicated plots of 54 F_1 hybrids having Anza as one parent were fall-planted in the field in 1970. Individual plants of the parents and F_1's were scored visually for BYDV after heading. The F_1's generally appeared to have resistance similar to Anza. The weighted mean F_1 score was compared to midparent values for all crosses. Figure 1 shows the frequency distribution for midparent deviations. Most F_1's showed negative values indicatative of dominance for resistance obtained from the Anza parent.

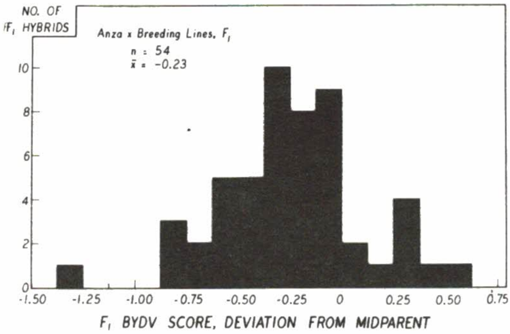

Figure 1. Frequency distribution of mid-parent deviations for BYDV reaction of 54 F_1 hybrids, all involving Anza. The other parents were breeding lines and varieties that had wide variation in BYDV reaction. Negative scores indicate that the F_1 approached the more resistant parent (Anza).

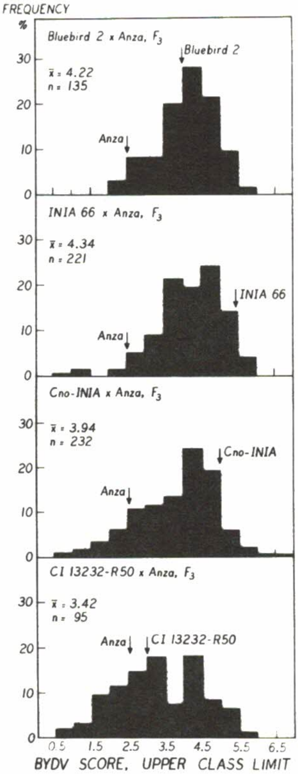

Figure 2. Frequency distributions for mean score of F_3 lines of four crosses. Low score indicates low symptom expression on a scale of 0 to 9. Parental mean scores are indicated by arrows.

Four crosses were evaluated in the spring-planted BYDV nursery in 1972. Two replicates of single-row plots of each F_3 line were scored visually on a plot basis. Frequency distributions for the mean BYDV scores are shown in Figure 2. The crosses INIA 66 and CNO-INIA with Anza show limited variation beyond the parental values, whereas the two remaining crosses show substantial transgressive segregation. With Bluebird 2 the transgression is mainly toward greater susceptibility, but in the cross with CI 13232-R50 transgression is seen for both resistance and susceptibility, with obvious bimodality of the distribution. CI 13232-R50 is one of the tolerant selections from the cross CI 13232 x Ramona 50. Since both parents have similar disease reactions and transgressive segregation was observed, it appears that different genes for resistance are present in the two parents. No interpretation of the bimodal distribution on the basis of a small number of genes will be attempted without further investigation.

Significant genetic variability was observed in all four crosses (Table 1). Heritability was moderately low (0.24 to 0.37), which no doubt reflects rather large environmental variation in the visual scoring system, being based on only two replicates. It is unusual that the largest genetic variance component was found in the resistant x resistant cross. This is encouraging because it may be possible to select resistant lines that are much better than either parent.

Table 1. Genetic and environmental variance components and heritability of BYDV reaction in F_3 lines, Davis, 1972

Hybrid P_1 x P_2	No. of F_3 lines	$\hat{\sigma}^2_g$	$\hat{\sigma}^2_e$	H
CNO-INIA x Anza	232	0.766**	1.284	0.37
Bluebird 2 x Anza	135	0.323**	1.049	0.24
INIA 66 x Anza	221	0.495**	1.128	0.30
CI 13232-Ram 50 x Anza	95[1]	0.889**	1.717	0.34

[1] 6 nonheading lines were excluded.

**p <.01.

From these preliminary results we conclude that there is genetic variability for resistance to BYDV in wheat. This resistance is inherited in a quantitative manner. Recurrent selection for resistance should be the most effective way to breed for BYDV resistance in wheat.

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