counted for by the wind.

## Conclusions

Stack surface temp increase as the burning rate increases on the large cone, return stack and auto clean stack heaters, but decrease on the spot heater. Surface temp on the spot heater are lower in general than the surface temp of the other three heaters tested. The position of the highest temp was near the bottom of the large cone, return stack and auto clean stack heaters but near the top on the spot heater.

Radiant fractions tend to be constant over the recommended range of burning rates. This implies that the radiant output of the heaters increases linearly with the burning rate. Radiant fractions tend to increase with the area of the heater stack. The radiant fraction of new shiny heaters was approx 30% lower than that of older and more oxidized heaters.

Although the technique used in this study underestimated the radiant output of the large cone, return stack and auto clean stack heaters, the observed radiant fractions for these heaters were greater than 20%. The total radiant fraction of the spot heater was overestimated due to the model flame method and was still less than 16%. At the higher burning rates, only 5% is due to the stack and the other 8% is contributed by the flame, i.e., the flame contributed as much as two-thirds of the radiant fraction.

The effect of wind speed on the radiant fraction was negligible in this study. However, there are theoretical arguments that would indicate that increasing wind speed should decrease the radiant fraction by decreasing the surface temp. Apparently this effect is negligible below the maximum of 2 m/sec wind speed observed in this study.

## Literature Cited

1. Aichelle, H. 1965. Frostschadensverhütung durch Geländeheizung. In Schnelle, F. (ed.) Frostschutz im pflanzenbau, Vol. 2, BLV Verlagsgesellschaft München Basal Wein.
2. Gerber, J. F. 1965. Performance characteristics of heating devices. Proc. Fla. State Hort. Soc. 78:78-83.
3. Hoffman, A. H. 1927. Laboratory tests on orchard heaters. Rpt. Calif. Agr. Expt. Sta. Davis.
4. Hottell, H. C. and F. P. Broughton. 1933. Determination of true temperature and total radiation from luminous gas flames. Ind. Eng. Chem. 4:166-175.
5. Kepner, R. A. 1951. Effectiveness of orchard heaters. Calif. Agr. Expt. Sta. Bul. 723.
6. Martsolf, J. D. 1974. Practical frost protection — frost incidence, site selection, control methods. Penn. Fruit News 53(5):15-30.
7. _____. 1976. Observed radiant loss from a heated orchard under frost conditions. J. Amer. Soc. Hort. Sci. 101:614-617.
8. McAdams, W. H. 1954. Heat transmission. McGraw-Hill, p. 60-62, 99-104, 472-476.
9. Reifsnyder, W. E. and H. W. Lull. 1965. Radiant energy in relation to forests. U. S. Dept. Agr. Forest Service Tech. Bul. 1344.
10. Truesdail Laboratories, Inc. 1967. Heat transmission study — oil fired orchard heater. Prepared for Spot Heaters, Inc., Sunnyside, Washington, by Truesdail Labs., Inc., Los Angeles, California.
11. Turrell, F. M. 1973. The science and technology of frost protection. p. 338-446, 505-558. In Reuther, (ed.) The citrus industry, Vol. 3, Rev. Ed. Univ. of Calif. Press.
12. Valli, V. J. 1970. Basic principles of freeze occurrence and the prevention of freeze damage to crops. Proc. Fla. State Hort. Soc. 83:98-109.
13. Wiebelt, J. A. 1966. Engineering radiation heat transfer. p. 186-194. Hot, Rinehart, and Winston.
14. Wilson, E. B. and A. L. Jones. 1969. Orchard heater measurements. Wash. Agr. Expt. Sta. Cir. 511.

# Diallel Analysis of Sweet Potatoes for Resistance to Fusarium Wilt[1]

W. W. Collins[2]

Department of Plant Pathology, North Carolina State University, Raleigh, NC 27607

Additional index words. general combining ability, specific combining ability, heritability, Fusarium oxysporum f. sp. batatas, Ipomoea batatas

Abstract. Eight clones of sweet potato Ipomoea batatas L. were evaluated for resistance to the soil-borne pathogen Fusarium oxysporum (Schlecht) f. sp. batatas (Wr.) Snydor & Hanson and their reactions varied from extremely susceptible to extremely resistant. The 8 clones were used as parents in a modified diallel crossing design which generated 24 families each consisting of 10 full-sib clones. These 240 clones were evaluated for Fusarium wilt resistance in a randomized complete block design. They showed a gradation of resistance similar to the parents which is consistent with the theory of a quantitative mode of inheritance for this character. Diallel analysis of the 24 families revealed significant general and specific combining ability effects. Additive variance accounted for 87% of the total genetic variance while dominance variance accounted for only 13%. Broad-sense and narrow-sense heritabilities estimated for individuals and full-sib families from the diallel analysis were consistently greater than 70%. Narrow-sense heritability of individuals using the parent-offspring regression method was 50%. Results indicate that gains in Fusarium wilt resistance should be rapid and substantial in sweet potatoes.

Fusarium wilt in sweet potatoes is caused by Fusarium oxysporum f. sp. batatas. The fungus is a long-term soil inhabitor which forms persistent chlamydospores and colonizes roots of nonsuscepts also. It is difficult to eliminate from the soil through conventional crop rotation or practical chemical applications. In sweet potatoes, as in many other crops attacked by the fusarium wilt fungi, resistant cultivars have been the most successful means of control.

Presently, many sweet potato breeding programs endeavor to incorporate wilt resistance into new cultivars. Several studies have concluded that resistance is multifactorial and that variability is extensive due to the natural outcrossing behavior of

J. Amer. Soc. Hort. Sci. 102(2):109–111. 1977.

109

sweet potatoes, their hexaploid nature, and the resultant hetero-zygosity (4, 5, 10).

However, there is very little knowledge about the causal components of variance and heritability of fusarium wilt resistance. Heritability estimates have ranged from 33% (8) to 86% (7). Jones (7) found the additive variance component accounted for most of the genetic variance measured with a very small amount of non-additive variance. His is the only study of this aspect of fusarium wilt resistance in sweet potatoes.

This study was to evaluate fusarium wilt resistance of breeding lines from a large random-mating population and their progenies in a diallel design in an effort to estimate the genetic variance in the random-mating population from which the lines were obtained. The nature of this variance and of gene action was also studied. Heritability was estimated with specific application to selection programs for improving and maintaining a high level of fusarium wilt resistance.

## Materials and Methods

The population from which the parental clones were selected originated from plants selected by Dr. Alfred Jones, USDA, ARS, Southern Region, Charleston, S. C., for good seed set and diverse origins. For this study 14 sweet potato clones were initially selected from Jones' I/3 population. However, based on flowering performance in the field during the summer of 1972 and on preliminary crosses, the no. of parental clones was reduced to 8. The clones were tested and categorized for resistance to the wilt fungus using the inoculation procedure and wilt index described by Collins and Nielsen (1). The 8 clones represented a wide range of resistance to fusarium wilt and flowered profusely under North Carolina field conditions.

The modified diallel cross procedure (Model II, Method 4) of Griffing (3) was used. The 8 parental clones were crossed in all combinations ignoring reciprocals (p(p-1)/2 possible crosses). Ten seeds of each cross were obtained except for 4 crosses which were incompatible regardless of which clone was used as the seed parent.

The seeds were acid-scarified and germinated in flats containing a 2 soil:1 sand:1 peat moss mixture. When seedlings were 10-12 cm tall they were transplanted to separate 15-cm clay pots containing the same soil mixture. Stem cuttings from subsequent growth of each seedling served as material for all disease tests.

All plants established in clay pots were fertilized weekly with 50 − 100 ml of nutrient solution. The solution contained VHPF − 700 g (6% N, 25% P, 15% K; Miller Chemical and Fertilizer Corp., Hanover, PA), $KNO_3$ − 123 g, and $MgSO_4 \cdot 7H_2$) − 227 g in 83 liters of tap water. Slower growing plants were sometimes fertilized twice a week to insure sufficient plant material for each test. Plants were watered as needed.

Two pathogenic isolates of *F. oxysporum* f. sp. *batatas* were used to evaluate the resistance reactions of the parental clones and their progenies. The isolates were maintained on

Table 1. Mean fusarium wilt indexes of 8 parental clones and their progeny.

| Parent | Wilt index | Mean wilt index of progeny |
|---|---|---|
| 821 | 0.42 | 0.73 |
| 334 | 0.70 | 1.11 |
| 723 | 0.99 | 1.37 |
| 219 | 1.00 | 1.35 |
| 532 | 1.03 | 1.33 |
| 567 | 1.85 | 1.27 |
| 372 | 2.25 | 1.59 |
| 904 | 2.48 | 1.65 |
| x̄ | 1.34 | 1.32 |
| r | 0.82 | |

Table 2. Fusarium wilt indexes of 24 families generated in a diallel cross design.[z]

| Seed parent | Pollen parent | | | | | | |
|---|---|---|---|---|---|---|---|
| | 219 | 532 | 567 | 334 | 904 | 723 | 821 |
| 327 | 1.87 | 1.75 | 1.67 | 1.43 | 2.05 | 1.44 | 0.89 |
| 219 | | 1.64 | 1.00 | 0.70 | 2.04 | 1.73 | 0.51 |
| 532 | | | 1.29 | 1.00 | − | 1.69 | 0.63 |
| 567 | | | | − | 1.38 | 1.01 | − |
| 334 | | | | | 1.43 | 0.99 | − |
| 904 | | | | | | 2.07 | 0.96 |
| 723 | | | | | | | 0.63 |

[z]Overall mean based on 4 replications, 10 progeny clones per family.

Table 3. Analysis of variance of diallel cross design.

| Source | df | SS | MS |
|---|---|---|---|
| Mean | 1 | 167.86 | 167.86 |
| Replications | 3 | 3.04 | 1.01 |
| Treatments | 23 | | |
| GCA | 7 | 19.32 | 2.76** |
| SCA | 16 | 1.94 | 0.12** |
| Error | 69 | 2.84 | 0.04 |
| Total | 96 | 195.00 | |

**F significant at 1% level.

potato-dextrose-agar (PDA) slants. The methods of preparing inoculum, inoculating vine cuttings and indexing disease reactions were those described by Collins and Nielsen (1). The index used covered a range of 0.00 (extreme resistance) to 3.00 (extreme susceptibility).

In the progeny analysis the means of full-sib families for each of 4 replications were used as data entires in a computer program (DIALL) developed by Schaffer and Usanis (9) for the general least squares analysis of diallel designs. Due to greenhouse space limitations, the 4 replications occurred over time.

## Results

The parental clones represented a range of fusarium wilt resistance from a resistant index of 0.42 to 2.48 (Table 1) with progeny means between 0.73 and 1.65.

Full-sib family means ranged from 0.51 (family 219 × 821) to 2.07 (family 904 × 723) (Table 2). Families derived from parent clone 821 gave consistently low fusarium wilt index readings, always less than 1.00 even in crosses with the most susceptible parents. The correlation of parent and offspring means (r = 0.82) indicates that, on the average, resistant parents give more resistant than susceptible offspring and susceptible parents give more susceptible than resistant offspring.

A diallel analysis showed both general and specific combining ability to be significant at the 1% level (Table 3). GCA and SCA components of variance ($\sigma^2_{GCA}$, $\sigma^2_{SCA}$) were estimated from the expected mean squares of the analysis (Table 4). These variance components can be shown to have the following genetic expectations under the assumptions of diploid inheritance, no epis-

Table 4. Components of variance and standard deviations from diallel analysis.

| Component | Value | SD |
|---|---|---|
| $\sigma^2_{GCA}$ | .129 | .064 |
| $\sigma^2_{SCA}$ | .020 | .010 |
| $\sigma^2_e$ | .041 | .007 |

110

Table 5. Estimates of variances and heritabilities of full-sib families from diallel analysis.

| | |
|---|---|
| $\sigma^2_a$ | .517 |
| $\sigma^2_d$ | .080 |
| $\sigma^2_G$ (total) | .597 |
| $\sigma^2_G$ (FSF) | .278 |
| $\sigma^2_P$ (FSF) | .289 |
| $h^2_{FS}$ | .894 |
| $H_{FS}$ | .962 |

tasis, independent assortment, and Hardy-Weinberg equilibrium:

$$\sigma^2_{GCA} = \tfrac{1}{4}\, \sigma^2_a$$

$$\sigma^2_{SCA} + \tfrac{1}{4}\, \sigma^2_d$$

in which $\sigma^2_a$ denotes additive genetic variance and $\sigma^2_d$ denotes dominance variance. These variances and the estimate of environmental variance were used to estimate genotypic variance, phenotypic variance, broad-sense heritability, and narrow-sense heritability of full-sib family means (Table 5). Broad-sense (H = .962) and narrow-sense ($h^2$ = .894) heritabilities of full-sib family means are exceptionally high and indicate that progress toward disease resistance through full-sib family selection should be rapid.

Individual heritabilities were estimated from the diallel analysis data and parent offspring regression (2) which also gave an additional estimate of additive genetic variance. Using the first method (diallel analysis) $\sigma^2_a$ was 0.517 and $h^2$ 0.714; using the parent-offspring regression method, $\sigma^2_a$ was estimated as 0.356 and $h^2$ as 0.503. The parent-offspring regression method is probably the more reliable method of estimating individual $h^2$ because $\sigma^2_a$ from the diallel analysis may be biased upward slightly. The fact that all the progenies were grown and evaluated at the same time led to a common environmental variance component which was reflected in $\sigma^2_a$. This component, however, was not present in the parent offspring regression method since parents and offspring were grown and tested separately.

### Discussion

Jones (6) has shown that chromosomal pairing in the majority of sweet potatoes occurs in a regular bivalent manner. The interpretation of the results of these studies is based upon the assumption that meiosis occurs in a regular diploid fashion.

The diallel analysis indicates that genetic variation for fusarium wilt resistance in sweet potatoes is mainly due to additive effects of genes ($\sigma^2_a$) which accounted for 87% of the total genetic variance (Table 5). This suggests that attempts to identify heterotic combinations are unnecessary. The dominance component of genetic variance ($\sigma^2_d$) was also significant in this study, but could be due to epistasis since one of the assumptions made in analyzing the results was that of no epistasis. If epistatic effects do exist, then the dominance variance component will reflect those effects as well as dominance variance. Further studies should be performed using designs to estimate epistatic variation in sweet potato to determine the magnitude and the effect on heritabilities of fusarium wilt resistance and other characters. Should epistatic variation prove to be significant, it could be very valuable in a sweet potato breeding program due to the asexual propagation of the plants.

Estimates of H and $h^2$ of full-sib families are high indicating that full-sib family selection might be valuable in a breeding program. Several family means in this study were below 1.00, and genetic advance would progress rapidly with full-sib family selection. In addition variability would be maintained in the population due to within-family segregation. Thus, full-sib family selection could be used to maintain a genetically variable population with a high level of fusarium wilt resistance from which individuals could be removed at intervals for advanced testing for future release.

### Literature Cited

1. Collins, W. W. and L. W. Nielsen. 1976. Nature of Fusarium wilt resistance in sweet potatoes. *Phytopathology* 66:489-493.
2. Falconer, D. S. 1960. Introduction to quantitative genetics. Ronald Press, New York.
3. Griffing, B. 1956. Concept of general and specific combining ability in relation to diallel cross systems. *Austral. J. Biol. Sci.* 9:463-469.
4. Guilbeau, S., J. J. Mikell, and J. C. Miller. 1952. A study of the inheritance of Fusarium wilt resistance in sweet potatoes. *Proc. Assoc. S. Agr. Workers 40th Ann. Conv.* 107 [Abstr.]
5. Hughes, M. B., C. E. Steinbauer, M. T. Deonier, and H. B. Cordner. 1963. Preliminary studies on the inheritance of wilt resistance in the sweet potato. *Proc. Amer. Soc. Hort. Sci.* 83:623-628.
6. Jones, A. 1965. Cytological observations and fertility measurements of sweet potato (*Ipomoea batatas* (L.) Lam.). *Proc. Amer. Soc. Hort. Sci.* 86:527-537.
7. ————. 1969. Quantitative inheritance of Fusarium wilt resistance in sweet potatoes. *J. Amer. Soc. Hort. Sci.* 94:207-208.
8. Kakar, R. S., Teme. P. Hernandez, T. Hernandez, and J. C. Miller. 1966. Inheritance of Fusarium wilt resistance in sweet potato, *Ipomoea batatas*. *Proc. Assoc. S. Agr. Workers 63rd. Ann. Conv.* 213-214 [Abstr.]
9. Schaffer, H. E. and R. A. Usanis. 1969. General least squares analysis of diallel experiments. A computer program – DIALL. Genetics Dept., Res. Rpt. 1., N. C. State University, Raleigh, N. C.
10. Struble, F. B., L. S. Morrison, and H. B. Cordner. 1966. Inheritance of resistance to stem rot and root-knot nematodes in sweet potato. *Phytopathology* 56:1217-1219.

J. Amer. Soc. Hort. Sci. 102(2):109–111. 1977.

111