Peanut Science (1980) 7, 13-14

Inheritance of a Necrotic-etch Leaf Disease in Peanuts[1]

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ABSTRACT

Little is known about the genetic control of diseases of the cultivated peanut, *Arachis hypogaea* L., and data on qualitatively inherited foliar maladies are rare. This paper reports the mode of inheritance of a disease of unknown cause. A necrotic-etch leaf condition, first observed in 1962, lacks distinctiveness or uniformity of affected areas, but is easily distinguished from foliar diseases caused by known pathogens. Affected plants breed true for the disorder. Attempts to isolate a causal agent have been unsuccessful.

Necrotic-etch leaf is inherited as a qualitatively controlled recessive trait. F_1 plants are normal. F_2 data for 3338 plants (3155 normal : 183 necrotic) in 44 progenies from 5 cross combinations gave good fits to the digenic 15:1 model, indicating duplicate, unlinked loci.

Gene symbols Ne_1 ne_1 Ne_2 ne_2 are proposed.

Key Words: Genetic-Disease, Arachis-Hypogaea, Groundnut, Genetic-Ratio, Non-Pathogenic.

Investigations of the qualitative and quantitative aspects of peanut (*Arachis hypogaea* L.) disease inheritance have appeared only rarely (3). No qualitative genetic data support the occasional claims for resistance to diseases of fungal origin enumerated in the most recent literature review (1). This paper describes and reports inheritance data for a peanut disease of undetermined cause.

The necrotic-etch leaf disease apparently arose spontaneously. Symptomswere first observed (2) in 1962 on a few plants in a nursery plot of the 'Jenkins Jumbo' peanut — a pure line that had been grown at the Coastal Plain Station since 1947 (5). Symptoms generally do not appear on the initial 12-20 leaves, but thereafter all newly expanded leaves show characteristic lesions (Fig. 1). When first visible, the lesion encompasses 3-5 cells with the direct center appearing to be a tertiary vein. A clorotic zone extends outward another 3-5 cells. Within two days, the necrotic area approximately doubles, advancing more rapidly through the secondary and tertiary veins than through the interveinal areas. The irregular zonate patterns, up to 0.5 x 1.0 cm in size, frequently appear directly opposite each other on either side of the leaflet mid-vein (D. K. Bell, *personal comm.).*

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When symptoms are first observed, no fungi or bacteria can be isolated from the primary spots, thus indicating the possibility of a non-pathogenic causal agent. No sign of insect puncture was seen at 400X magnification. Attempts to transmit the disease mechanically were unsuccessful (C. W. Kuhn, *personal comm.).* None of the described diseases of peanut incited by fungi, bacteria, viruses, or nematodes, match the symptoms of the disorder. The incitant of necrotic etch has not been established despite repeated attempts to isolate or identify an infectious or noninfectious cause.

However, since hybridizations give evidence that the disease expression is under genic control, the term "genetic disease" is an appropriate designation for the necrotic-etch leaf malady. Genetic disease is defined as a disorder identified as having a genetic basis.

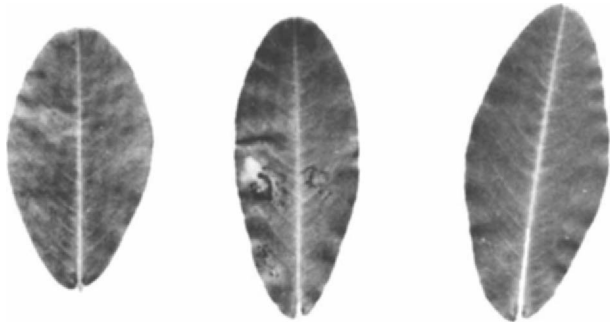

Fig. 1. Necrotic-etch leaf disease symptoms: *Left,* chlorotic zone surrounding a lesion in early stage of development; *center,* advanced stage; *right,* normal leaf.

Materials and Methods

Diseased (JD) plants and their disease-free (JF) sibs were isolated, progeny tested, and further maintained by natural self fertilization. Single plant isolates from the necrotic-etch line were crossed with 5 other inbreds to evaluate character inheritance and expression in diverse genotypic backgrounds.

Artificial cross pollinations were made in the greenhouse using standard procedures (6). F_1 plants were spaced individually in 90 x 90 cm plots. F_2 plots varied in length with the quantity of seed available from each F_1, and plants were spaced 30 x 90 cm. Adjacent plots contained self-pollinated parental genotypes. The nurseries were grown under semi-commercial production management at the agronomy research farm near Tifton.

Plants were classified between mid-season and harvest. Analyses for the two-class phenotypic segregation were by chi-square criteria for each of the 44 independent progenies. For each of the 5 crosses 3 additional chi-squares per combination — the sum, the pooled, and the homogeneity — were appropriate measures of variance. Finally, a further summation of the 5 families gave 3 additional chi-squares (making 62 tests of the 44 progenies) to evaluate goodness-of-fit for the entire F_2 population.

Results and Discussion

The 5 combinations from artificial cross pollinations gave 44 F_1 plants. Symptomless phenotypic appearance of the 44 plants from crosses of normal (JF) with necrotic-etch (JD) genotypes indicated dominant inheritance for the nondiseased condition. Observed F_2 segregation for the individual progenies fit the expectation for the 15:1 dihybrid ratio : 42 of the 44 chi-squares were above the 5% probability level, and the remaining 2 progenies had 4% and 3% probabilities, respectively. To conserve space, individual progeny chi-squares are ommitted.

When F_2 distribution data for the 5 different cross combinations were examined, the absence of significant deviation within the 5 progeny sets and the homogeneity of both within-cross and combined-cross population ratios confirmed inheritance of the necrotic-etch condition as a digenic recessive (Table 1).

Table 1. Necrotic-etch leaf disease inheritance in crosses of diseased with non-diseased peanut genotypes, and chi-square tests for goodness-of-fit to the 15:1 F_2 ratio.

Cross No.	Pedigree[1/] ♀ ♂	Progenies No.	Leaf disease None No.	Leaf disease Etch No.	Chi-squares and Probabilities[2/] Sum χ^2	P ca.	Pool χ^2	P ca.	Homo. χ^2	P ca.
C 205	JD X JF 12	13	467	27	4.723	.98	0.519	.48	4.204	.98
C 207	JD X WG-II	3	126	11	5.830	.13	0.740	.41	5.090	.08
C 210	JD X Alba	5	569	25	5.042	.42	4.224	.04	0.818	.93
C 212	JD X T 1860	12	868	52	18.788	.10	0.561	.47	18.227	.07
C 215	JD X T 1919	11	1125	68	11.387	.42	0.616	.45	10.771	.38
Five Crosses		44	3155	183	45.770	.45	3.357	.07	42.413	.50
D.F. for χ^2					44		1		43	

1/ Pedigree abbreviations: J = 'Jenkins Jumbo' parental line; D and F designate diseased and non-diseased genotypes selected from Jenkins Jumbo. The other four parents are unrelated genetic stocks in the USDA-SEA-AR nursery.

2/ Chi-squares and probabilities for the 44 individual progenies are omitted to conserve space.

The pooled chi-squares measure the goodness-of-fit to a theoretical binomial proportion. One of the 5 within-cross pooled chi-squares, 4.224, had a probability near the 4% level. In this cross, C 210, the nondiseased parent grows slowly and matures *ca.* 180 days after planting. The 5 progenies in C 210 had a cumulative deficit of only 12 recessive plants. Field experience has shown that the deviations can be partly explained by the weakened condition of plants in the double recessive class because of reduced growth. Since C 210 also segregated for wide differences in plant duration, early maturing diseased plants were more likely to die than their nondiseased sibs. When chi-square is computed for the pooled sample of the 44 progenies comprising all 5 cross combinations, its value of 3.357 (P = *ca.* .07) reflects the consistent deficit of the diseased phenotype.

In addition to the 15:1 ratio, the data also gave acceptable fits to the 243 : 13 tetragenic ratio. However, the homogeneity chi-squres were invariably larger for the latter ratio. Furthermore, none of the F_3 populations suggest anything other than the digenic model.

The genetic behavior suggests that necrotic-etch leaf is conditioned by two duplicate unlinked loci. Gene symbols Ne_1 and Ne_2 are proposed for normal and ne_1 and ne_2 for the necrotic-etch leaf disease.

Under the present circumstances the necrotic-etch leaf disease is of no apparent economic importance. Nevertheless. the history of plant diseases often shows that once minor diseases may become of major significance almost without warning. The peanut is host to a wide array of pathogens and is genetically vulnerable to destruction by an epidemic (4). Further research is warranted.

Acknowledgment

D. K. Bell, Dept. of Plant Pathology, Coastal Plain Stn., Tifton, and C. W. Kuhn, Dept. of Plant Pathology and Plant Genetics, Univ. Georgia, Athens, assisted in describing the apparent non-pathogenicity and expressions of this genetic disease.

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Accepted February 9, 1980