Inheritance of Resistance to Anthracnose in Blue Lupines

Lupinus angustifolius L.¹

Ian Forbes, Jr. and Homer D. Wells²

BLUE lupines (Lupinus angustifolius L.) are cultivated in the eastern gulf coast area of the United States as a winter annual leguminous cover crop or temporary pasture. Anthracnose of blue lupines, caused by Glomerella cingulata (Ston.) Spauld. & Schrenk, results in serious losses of forage and seeds in southern Georgia, southern Alabama, and northern Florida in wet seasons. Weimer (7) published a comprehensive account of the disease and the pathogen. The fungus attacks the lupine plant at all stages of growth. It kills plants by girdling the main stem and reduces seed production and quality by infecting the pods and seeds. Weimer (7) found a high degree of seedling resistance to anthracnose among wild bitter blue lupine introductions from Portugal. Blue lupine was found to be almost entirely self-pollinated at Gainesville, Florida (6), and entirely so at Tifton, Georgia (1).

The present study was concerned with the inheritance of resistance to anthracnose found by Weimer in a wild bitter blue lupine introduction, P.I. 168535, from Portugal. Bitter blue lupines contain toxic concentrations of alkaloid which make them useless for pastures. Sweet (low-alkaloid) varieties, Borre and Blanco blue lupine, in use for pasture in the United States, owe their low alkaloid content to the presence of the recessive pair of genes, inc inc, found by Von Sengbusch (4) in his strain 411.

MATERIALS AND METHODS

A description of parents used in this study follows. P.I. 168535, the introduction from Portugal, found by Weimer to contain anthracnose-resistant plants, is bitter, hard-seeded, and blue-flowered. A white-flowered and white-seeded bitter selection (WFB), which is anthracnose-susceptible and soft-seeded, originated as a mutant in common bitter blue lupine grown in Georgia. Blanco blue lupine (2) originated from WFB×Borre, a cross made in 1952, and is homozygous for sweetness, soft seeds, white flowers and seeds, and anthracnose susceptibility. Selection 54–202–1–C, which arose from the cross WFB×P.I. 168535, is homozygous for anthracnose resistance, bitterness, white flowers and seeds, and soft seeds.

In 1952, the cross WFB×P.I. 168535 was made for the purpose of a preliminary study of inheritance of anthracnose resistance, and to obtain breeding lines homozygous for anthracnose resistance, soft seeds, and the genetic marker white flowers and seeds. In 1954, F₂ plants having soft seeds and white flowers and seeds were selected. F₂ and F₃ families from these F₂ selections were tested for disease reactions in the winters of 1955 and 1956 by spraying greenhouse-grown seedlings with aqueous suspensions of the fungus mycelium and conidia and then holding them in a moist chamber for 48 hours before removing them to greenhouse benches. The plants were rated for disease reactions 14 days after inoculation. Data obtained from these preliminary attempts to inoculate and screen lupine populations for disease resistance were useless for determining the mode of inheritance of resistance because large numbers of susceptible plants escaped infection. The fact that susceptible plants included with each batch of plants following factors were apparently responsible for results obtained: (1) Excessive output of the fog moist chamber sometimes washed the inoculum from the fog machine from becoming moist enough for infection. (2) Large size of the moist chamber prevented some seedlings being shielded from the inoculating greenhouse-grown blue lupine plants.

Although reliable genetic data were not obtained in the preliminary study, some F₂ plants homozygous for anthracnose resistance, soft seeds, and white flowers and seeds were obtained. One of these plants, 54–202–1–C, was used as Blanco lupine in 1956. In 1957, 1958, and 1959 F₂ and F₃ generations of this cross were classified for disease reaction, and the F₂ and F₃ were classified for bitterness, soft seeds, and the genetic marker white flowers and seeds. In 1959, F₃ plants were grown one seedling per pot to give the greatest precision in inoculation. Fa families from the resistant F₂ survivors were grown (4 seedlings per pot) to provide 32 control plants of each for every inoculation series. The F₂ plants, 2 weeks to 2 months) were placed in a moist chamber and greenhouse benches. Disease-reaction ratings were made 14 days after inoculation. The apparently disease-resistant survivors from each plant were grown (4 seedlings per pot) to provide 32 control plants of each for every inoculation series. The F₃ plants were inoculated in the same manner as the F₂ plants, except that (1) F₃ plants were grown one seedling per pot to give the greatest precision in inoculation, (2) F₃ families from the resistant F₂ survivors were grown (4 seedlings per pot) to provide 32 control plants of each for every inoculation series, (3) F₃ plants were transplanted separately to Petri dishes containing V-8 juice agar. After growing in the laboratory at room temperature for 2 weeks, they were ground for 30 seconds in 5 ml of water and then diluted 1:100 with water. Absence of reaction indicated control plant reactions indicated that inoculation had not been effective. Previous defects in the moist chamber and greenhouse, large size of the moist chamber prevented some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum.

Disease-reaction ratings were made 14 days after inoculation. The apparently disease-resistant survivors from each plant were grown (4 seedlings per pot) to provide 32 control plants of each for every inoculation series. The F₃ plants were inoculated in the same manner as the F₂ plants, except that (1) F₃ plants were grown one seedling per pot to give the greatest precision in inoculation, (2) F₃ families from the resistant F₂ survivors were grown (4 seedlings per pot) to provide 32 control plants of each for every inoculation series, (3) F₃ plants were transplanted separately to Petri dishes containing V-8 juice agar. After growing in the laboratory at room temperature for 2 weeks, they were ground for 30 seconds in 5 ml of water and then diluted 1:100 with water. Absence of reaction indicated control plant reactions indicated that inoculation had not been effective. Previous defects in the moist chamber and greenhouse, large size of the moist chamber prevented some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum.

The surviving disease-resistant plants were handled in the pots outdoors during the daytime for a week, transplanted to field nurseries, where they were protected from freezing by covering with 5-quart cans. In the field, the plants were tested in the field for alkaloid content, the method used by European workers, 3) the plants were transplanted separately to Petri dishes containing V-8 juice agar. After growing in the laboratory at room temperature for 2 weeks, they were ground for 30 seconds in 5 ml of water and then diluted 1:100 with water. Absence of reaction indicated control plant reactions indicated that inoculation had not been effective. Previous defects in the moist chamber and greenhouse, large size of the moist chamber prevented some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum.

The surviving disease-resistant plants were handled in the pots outdoors during the daytime for a week, transplanted to field nurseries, where they were protected from freezing by covering with 5-quart cans. In the field, the plants were tested in the field for alkaloid content, the method used by European workers, 3) the plants were transplanted separately to Petri dishes containing V-8 juice agar. After growing in the laboratory at room temperature for 2 weeks, they were ground for 30 seconds in 5 ml of water and then diluted 1:100 with water. Absence of reaction indicated control plant reactions indicated that inoculation had not been effective. Previous defects in the moist chamber and greenhouse, large size of the moist chamber prevented some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum.

The surviving disease-resistant plants were handled in the pots outdoors during the daytime for a week, transplanted to field nurseries, where they were protected from freezing by covering with 5-quart cans. In the field, the plants were tested in the field for alkaloid content, the method used by European workers, 3) the plants were transplanted separately to Petri dishes containing V-8 juice agar. After growing in the laboratory at room temperature for 2 weeks, they were ground for 30 seconds in 5 ml of water and then diluted 1:100 with water. Absence of reaction indicated control plant reactions indicated that inoculation had not been effective. Previous defects in the moist chamber and greenhouse, large size of the moist chamber prevented some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum.

The surviving disease-resistant plants were handled in the pots outdoors during the daytime for a week, transplanted to field nurseries, where they were protected from freezing by covering with 5-quart cans. In the field, the plants were tested in the field for alkaloid content, the method used by European workers, 3) the plants were transplanted separately to Petri dishes containing V-8 juice agar. After growing in the laboratory at room temperature for 2 weeks, they were ground for 30 seconds in 5 ml of water and then diluted 1:100 with water. Absence of reaction indicated control plant reactions indicated that inoculation had not been effective. Previous defects in the moist chamber and greenhouse, large size of the moist chamber prevented some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum, large size of the moist chamber prevented some seedlings being shielded from the inoculum, and some seedlings being shielded from the inoculum.