Table 5

F_2 segregations for an expected 15:1 ratio involving the L62-904 gene and various other genes

Cross	Race	Resistant O	Resistant E	Susceptible O	Susceptible E	Chi-square	P
Rps$_1$							
L62-904 x OX708 + OX900	1	107	108.8	9	7.2	0.25	0.60-0.50
Rps$_3$							
L62-904 x PRX8-5	4	56	55.3	3	3.7	0.12	0.80-0.70
Rps$_4$							
L62-904 x PI86,050*	4	64	67.5	8	4.5	2.13	0.20-0.10
Altona - Rps							
L62-904 x OX693	4	105	103.1	5	6.9	0.30	0.60-0.50
Kingwa - Rps							
L62-904 x OX696	3,4,5	54	54.4	4	3.6	0.05	0.90-0.80

*F_2 seedlings inoculated as described by Ward et al. (1979); other crosses were F_3 seedling tests of F_2 plants using hypocotyl wounding/mycelium insertion (Buzzell et al., 1977).

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1) An allelism study of the inheritance of the lack of soybean lectin in five soybean lines.

Pull et al. (1978) found five soybean lines ('Columbia', 'Norredo', 'Sooty', T102 and 'Wilson-5') lacking the 120,000 dalton seed lectin, also called soybean lectin (SBL). Orf et al. (1978) established that the lack of SBL is inherited as a simple recessive, *le le*. The homozygous dominant

(*Le Le*) and heterozygous (*Le le*) condition result in the presence of SBL. In the above inheritance study only one of the five lines, T102, was used to determine the inheritance of SBL. The study reported herein was conducted to determine whether *le le* is allelic in the five lines lacking SBL.

A diallele set of crosses was made among the five lines Columbia, Norredo, Sooty, T102 and Wilson-5. Seeds were checked for the presence of SBL using the Ouchterlony (1948) double diffusion technique with SBL antiserum (Orf, 1979). Only a small chip of each seed is necessary for this nondestructive method. F_2 seeds were harvested from F_1 plants and 20 seeds from each cross were checked for SBL by the Ouchterlony method. No F_2 seeds were available for the cross T102 x Columbia.

All F_1 and F_2 seeds lacked SBL. The results indicate that *le le* is allelic in the five lines lacking SBL.

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2) Soybean seed β-amylase variants

Hildebrand and Hymowitz (1980a) reported that two soybean genotypes were found that lack detectable seed β-amylase activity. The cultivar 'Chestnut' produces an inactive β-amylase protein, Sp^{an} (Hildebrand and Hymowitz, 1980b); 'Altona' is a mixture of genotypes that have a β-amylase protein of normal activity (Sp_1^{b}) or lack it entirely (sp_1) (Hildebrand and Hymowitz, 1980b).

Chestnut was selected from 'Habaro' and introduced into the U.S. as PI 20,405 in 1906 from Kharbarovsk, USSR (Hymowitz et al., 1977). All 30 seeds of Habaro we tested for β-amylase were found to have normal β-amylase activity.

Moreover, there are vast differences between seed characteristics, plant growth habit, plant morphology, maturity, etc. between Chestnut and Habaro. Most likely Chestnut was selected from alien genetic material in the Habaro seed lot (R.L. Bernard, personal communication). Therefore, the origin of Chestnut apparently is unknown.

Altona was selected from the cross PI 194,654 x 'Flambeau' (Hymowitz et al., 1977). Flambeau was introduced into the U.S. in 1934 from the USSR (Hymowitz et al., 1977) and PI 194,654 was introduced into the U.S. from Sweden (Bernard, 1965). To determine if either of the parents of Altona was the source of the sp_1 alleles, 10 seeds of both Flambeau and PI 194,654 were tested for β-amylase activity. All seeds of both lines had normal β-amylase activity.

Altona was composited in the F_5 and it traces back to a single F_4 plant (Bernard and Lindahl, 1970). The most likely explanation for the situation in Altona is that a mutation occurred at the β-amylase locus in a F_4 seed on a F_3 plant, resulting in a heterozygous F_4 plant ($Sp_1^{\ b}\ sp_1$) genotype. Since both Altona genotypes probably trace back to a single F_4 plant, they represent near isogenic lines with about 94% of the loci having identical alleles. This is consistent with the lack of any differences in morphology or yield of these two genotypes in observation nurseries in Minnesota (J. W. Lambert, personal communication).

The lack of β-amylase activity in certain genotypes of Altona perhaps is due to amylase inhibitors (Jaffe and Lette, 1968). However, we found that mixing equal volumes of pH 5.0 acetate extracts of Altona ($Sp_1^{\ b}$) with extracts from Altona (sp_1) and incubating the mixtures at 4C for 24 hours gave an intermediate level of β-amylase activity. Also, the fact that sp_1 is recessive to $Sp_1^{\ b}$ indicates that sp_1 probably is due to mutation resulting in the lack of synthesis of the β-amylase protein (Hildebrand and Hymowitz, 1980b).

We have found no marked differences in chemical composition or carbohydrate metabolism in developing or germinating seeds of the soybean cultivars 'Williams' ($Sp_1^{\ b}$), Chestnut ($Sp_1^{\ an}$), Altona ($Sp_1^{\ b}$) and Altona (sp_1) (Hildebrand and Hymowitz, n.d.). Perhaps β-amylase in soybeans is just a storage protein or has some survival value such as conferring a greater level of resistance to a specific pest or pathogen.

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