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Selection for increased percentage phaseolin in common bean

2. Changes in frequency of seed protein alleles with S_1 family recurrent selection

D.E. Delaney* and F.A. Bliss,*****

Department of Horticulture, University of Wisconsin, Madison, WI 53706, USA

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Summary. Recurrent selection methods are designed to increase the frequency of favorable alleles within a population(s) with each cycle of selection. Yet it is likely that different methods will change allele frequencies at different rates or may act on different alleles. To investigate the ability of S_1 family selection to shift the frequencies of favorable alleles within a population of *Phaseolus vulgaris* (L.), we examined the changes in frequencies of six alleles (*Phas*^{S}, *Phas*^{C}, *Phas*^{T}, *phas*$^-$, *lec*$^-$, and *Arc1*$^+$) that affect the amount of phaseolin accumulated in seeds, over three cycles of selection for increased percentage phaseolin (PPS). The frequency of alleles *Phas*^{C} and *lec*$^-$, both of which have positive effects on percentage phaseolin, increased with selection while the frequencies of *phas*$^-$ and *Arc1*$^+$, which have strong negative effects, decreased. The frequencies of the *Phas*^{S} and *Phas*^{T} alleles showed no linear trends with selection, indicating that the frequency changes may be due to random drift and not to the selection procedure. The proportion of the phenotypic variation (R^2) for percentage phaseolin that was explained by each of the alleles, and by all the alleles combined, changed with each cycle of selection. In most cases the change resulted in a decrease in the R^2 value. In this population, S_1 family selection was effective at increasing the frequencies of all favorable alleles except *Phas*^{T}, and rapidly decreased the frequencies of deleterious alleles.

Key words: Seed storage proteins – Recurrent selection – Frequency of alleles – *Phaseolus vulgaris* (L.) – SDS-PAGE

* *Current address*: Department of Plant Pathology, Kansas State University, Manhattan, KS 66506, USA
** *Current address*: Department of Pomology, University of California, Davis, CA 95616, USA
*** To whom correspondence should be sent

Introduction

Quantitative traits often are said to be controlled by many genes, each with an individual effect too small to be measured conveniently under usual experimental conditions. Contrary to this assumption, there are numerous examples of single genes having rather large effects on quantitative expression. It is more likely that genes affecting quantitative traits have a range of effects from large to small, depending on the background in which they are expressed. The continuous nature of quantitative inheritance makes studying individual alleles difficult. Theoretically, alleles having positive effects on the trait of interest should increase in frequency with selection and those having negative effects should decrease. Also, the frequency of alleles with large effects should increase or decrease faster than the frequency of alleles with relatively small effects. Without easily identifiable alleles, it is difficult to document changes in frequency.

Stuber and Moll (1972) and Stuber et al. (1980) observed enzyme allele frequency changes that were associated with selection for increased yield in maize. These changes were found to be too large to be due to random drift alone. In another study, Edwards et al. (1987) and Stuber et al. (1987) used variation at isozyme loci in F_2 populations to establish linkage relationships between 17–20 isozyme alleles and 25 yield-related traits. From those data they were able to map regions of the genome where loci affecting yield may be located, determine the magnitude of effects of quantitative trait loci associated with individual isozyme alleles, and determine type of gene action involved.

In a simulation study comparing changes in gene frequency associated with three methods of selection (mass, modified ear-to-row, and S_1 family selection), Choo and Kannenburg (1979) found that changes in gene frequen-

cy were greatest with S_1 family selection, regardless of initial frequency and gene effect. However, more desirable alleles were lost under S_1 family selection than with the other two methods. The number of genes lost depended primarily on initial gene frequency, but also was affected by gene action, magnitude of gene effect, heritability, and selection intensity.

There is substantial genetic variation for seed storage protein concentration in many crops. The process of accumulation and deposition of these proteins is well known, particularly in the cereals and legumes, making them good subjects for detailed genetic study. Mutants have been identified in cereals that have qualitative effects on the production of different storage protein fractions (Nelson 1979; Doll 1983; Singh and Axtell 1973). Qualitative variation for the production of seed storage proteins is present also in common bean. In backcross-derived lines with *S*-, *T*-, and *C*-type phaseolin, *T*- and *C*-types were significantly higher in percentage phaseolin and milligrams of phaseolin per seed than *S*-types (Hartana 1986). The absence of lectin (lec^-) increases the amount of phaseolin that is produced in all three phaseolin types (Osborn and Bliss 1985; Hartana 1986). When lectin is not synthesized, the increase in phaseolin over compensates for the lack of lectin. When the novel protein arcelin is produced ($Arc1^+$), the amount of phaseolin is reduced by over 50% (Romero-Andreas et al. 1986). Lines homozygous for a recessive phaseolin-deficient allele ($phas^-$), isolated from the *P. coccineus* cultivar 'Mexican Red Runner', produce no detectable amounts of phaseolin. Sullivan and Bliss (1983 b) identified several lines among three populations that had either discretely higher phaseolin concentration (milligrams per gram of flour) or milligrams of phaseolin per seed. They suggested the differences were due to one or a few genes.

The interrelationships among the alleles for phaseolin, lectin, and arcelin that influence the accumulation of phaseolin make it a good system for studying quantitative expression, because the effects of alleles both at the locus itself and at other unlinked loci can be examined. The objective of this study was to examine the effects of selection on alleles having different initial frequencies, magnitudes, and directional (positive or negative) effects on phaseolin. We also wanted to determine what effects the selection system may have on changes in allele frequency and accumulation of favorable alleles.

Materials and methods

Description of parents

Seven breeding lines (2-4-1, 6-30, PP11-37, 809598-3, L12-56, SARC1-7, and BMC 3522) were used as parents of the base population. All lines, except BMC 3522, have the cultivar 'Sanilac' in their pedigree and have similar seed type. Among the seven lines are four different alleles for phaseolin type ($Phas^S$, $Phas^C$, $Phas^T$, and $phas^-$) and two alleles, lectin-deficient (lec^-) and arcelin-1 ($Arc1^+$), that are at loci not linked to phaseolin but that are linked in coupling phase to each other (Osborn et al. 1986). Whenever arcelin-1 is produced, lectin is produced also. No $Arc1^+$ $Arc1^+/lec^-$ lec^- recombinants have yet been identified. The line 2-4-1 originated from a recurrent selection population in which selection had been practiced for both high protein and high yield (Sullivan and Bliss 1983 a). The seeds contain *S*-type phaseolin but have the highest percentage phaseolin of all the parents. The line 6-30 also contains *S*-type phaseolin and was selected from an inbred-backcross population because it was discretely higher in both milligrams of phaseolin per seed and milligrams of nonphaseolin protein per seed (Sullivan and Bliss 1983 b). It is not known whether the favorable gene(s) for increased phaseolin these lines carry are linked to the $Phas^S$ allele. The mean percentage phaseolin accumulation of each of the parental lines and the effects of each alleles relative to the standard cultivar 'Sanilac' were shown previously (Delaney and Bliss 1990).

Population development and selection scheme

The development of the base population and selection procedures were described in detail (Delaney and Bliss 1990) and will only be reviewed here. The base population was generated by two cycles of intercrossing among the parental lines, first using a half-diallel design and then a partial-diallel design. Selection was initiated in 1985 among the S_1 seeds of 95 double-cross families, from which the 20% (19 families) with the highest percentage phaseolin were selected. Remnant S_1 seeds of selected families were intercrossed using a chain cross design and then selfed. Replicated S_1 families were evaluated in the second and third cycles of selection and, again, the best 20% were selected (24 families in both cycles). The criterion for selection was percentage phaseolin (milligrams of phaseolin per 100 mg flour), which was determined by rocket immunoelectrophoresis (Laurell 1966; Sun et al. 1978), without regard to the presence or absence of particular alleles for the seed proteins.

Determination of allele frequency

Since the seven parental lines were highly inbred and homozygous for the six seed protein alleles, the allelic frequencies in the base population (C_0) were determined empirically from the double-cross pedigrees. The allelic frequencies in the three selected cycles (C_1, C_2, and C_3) were determined by SDS-polyacrylamide gel electrophoresis (SDS-PAGE). The procedure was that used by Laemmli (1970) as modified by Ma and Bliss (1978).

Sixty randomly chosen individual seeds from each of the selected families in each of the three cycles of selection were taken from a balanced composite of each S_1 family and screened for the presence or absence of six seed protein alleles ($Phas^S$, $Phas^C$, $Phas^T$, $phas^-$, lec^-, and $Arc1^+$). Seeds that were $Phas^C/Phas^T$ heterozygotes could be easily distinguished from $Phas^S$, $Phas^C$, and $Phas^T$ homozygotes and from $Phas^S/Phas^C$ and $Phas^S/Phas^T$ heterozygotes; however, it was difficult to distinguish $Phas^S/Phas^T$ heterozygotes from $Phas^S/Phas^C$ heterozygotes using this method (Fig. 1). Therefore, in families that were segregating for all three phaseolin types, seeds of heterozygotes were planted in the greenhouse and the plants were allowed to self-pollinate. At least ten seeds of each plant were then screened to determine the genotype of the parent. Seeds heterozygous for arcelin-1 had slightly darker phaseolin bands than did homozygotes for arcelin-1, and heterozygotes for $phas^-$ had slightly lighter phaseolin bands than seeds homozygous for phaseolin production. These distinctions may not be completely reliable, therefore the frequencies should be considered best estimates.

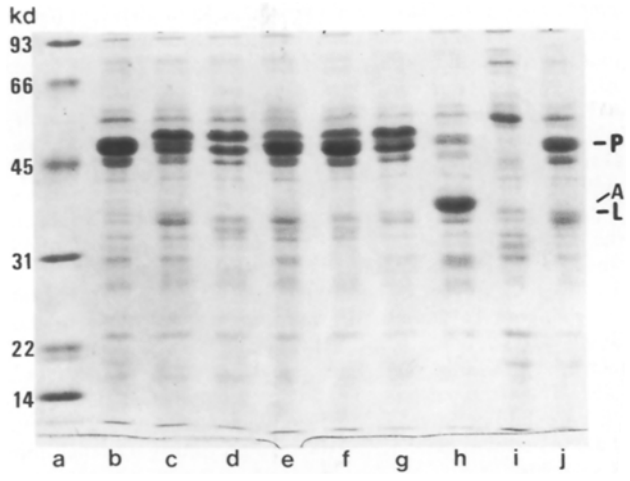

Fig. 1. SDS-PAGE of seed protein markers. *a* – Molecular weight markers (mol wt indicated to the *left* in kD); *b* – S-type phaseolin (*Phas*^{S}/*Phas*^{S}), lectin-deficient (*lec*$^-$/*lec*$^-$); *c* – *C*-type phaseolin (*Phas*^{C}/*Phas*^{C}), *S*-type lectin (*Lec*^{S}/*Lec*^{S}); *d* – *T*-type phaseolin (*Phas*^{T}/*Phas*^{T}), *T*-type lectin (*Lec*^{T}/*Lec*^{T}); *e* – *Phas*^{S}/*Phas*^{C}, *Lec*^{S}/*Lec*^{T}; *f* – *Phas*^{S}/*Phas*^{T}, *Lec*^{T}/*lec*$^-$; *g* – *Phas*^{C}/*Phas*^{T}, *Lec*^{S}/*lec*$^-$; *h* – *Phas*^{S}/*Phas*^{S}, *arcelin-1* (*Arc1*$^+$/*Arc1*$^+$); *i* – phaseolin-deficient (*phas*$^-$/*phas*$^-$), *lec*$^-$/*lec*$^-$; *j* – *Phas*^{S}/*phas*$^-$, *Lec*^{S}/*lec*$^-$. *P* = *phaseolin*, *L* = *lectin*, *A* = *arcelin-1*

The identification of double heterozygotes was not necessary because none of the selected families were segregating for both *Arc1*$^+$ and *phas*$^-$. Since arcelin-1 and lectin migrate to approximately the same position in SDS-PAGE, a haemagglutination assay was used to determine the presence or absence of lectin in seeds that produced arcelin-1 (Brown et al. 1981).

Statistical methods

Standard errors for the estimates of the frequency of each allele in each cycle were calculated using the variance among the frequency estimates for the individual selected families (Buri 1956). To determine whether the changes in allele frequency were due to random drift or to selection, a regression analysis similar to that described by Schaffer et al. (1977) and Wilson (1980) was used. The model accounts for two sources of sampling variation in selection experiments. The first is variation created by choosing a finite number of individuals as parents of the next cycle, i.e., random genetic drift. The second is the variation associated with the sample sizes of seeds or plants used for determination of the allele frequencies in each cycle.

In the analysis it was assumed that plants to be selfed were chosen randomly and independently from a random-mating population, so that the sampling of genes followed a binomial distribution. In this study, a sample of 60 seeds from each of the selected S_1 families generated from a random-mating population were randomly and independently chosen for analysis (a total of 1,140 seeds for C_1 and 1,440 seeds for C_2 and C_3 were screened). Since the selected families were used as parents for the next generation and the frequency of the alleles was determined for each, the allele frequency in each cycle was estimated with a high degree of precision. Therefore, the error due to sampling for analysis of allele frequency was assumed to be near zero. Thus, the only sampling variation of concern was that due to using a small number of selected S_1 families to form the next generation.

The analysis was performed on arcsin transformed data as suggested by Wilson (1980). The transformed allele frequencies were equal to:

$$2\sin^{-1}\sqrt{p_i}$$

where p_i is the allele frequency for the i^{th} cycle. This was modified to $2\sin^{-1}\sqrt{1/2n_i}$ for $p_i=0$, and $2\sin^{-1}\sqrt{1-1/2n_i}$ for $p_i=1$, where n_i is twice the number of seeds sampled in the i^{th} cycle. The allele frequency in the base population was known with certainty, therefore the model was modified by subtracting the allele frequency in C_0 from the frequency in each of the other cycles. This forced the regression through the point (C_0, p_0), similar to forcing a line through the origin (Steel and Torrie 1980).

The model assumes that all variation in allele frequency is due to random drift; thus, significant deviations from the model are interpreted as effects due to selection. The Chi-squared distribution was used to test the significance of deviations and linear and quadratic responses. Linear and quadratic trends were tested by fitting separate models to the data, since only three degrees of freedom were available.

The proportion of the variation for percentage phaseolin that was explained by each of the seed protein alleles was determined by calculating the coefficients of determination (R^2) due to regression of phenotypic values on allele frequencies in the selected families (Edwards et al. 1987). The R^2 values for models including main effects of each individual allele, and for multiple regression models including cumulative effects of all alleles, are presented for the parental lines and cycle 1 through cycle 3.

Results and discussion

Changes in frequency of seed protein alleles

The effect of S_1 family recurrent selection on changing the frequency of the seed protein alleles differed, depending on the allele. The frequency of the *Phas*^{S} allele increased initially from 0.574 in C_0 to 0.637 in C_1, but then decreased in C_2 and C_3 (0.559 and 0.461, respectively) (Table 1). Tests for deviations from a random drift model, however, were not significant (Table 2). This can be interpreted in two ways: the changes in allele frequency may actually be due only to random drift, or the models fit to the data may not be sensitive to a change of this kind.

The *Phas*^{C} and *Phas*^{T} phaseolin alleles started at the same initial frequencies and their effects on percentages phaseolin are of similar magnitude, yet the frequencies of the *Phas*^{C} allele increased 162% between C_0 and C_3 (from 0.147 to 0.385), while the frequencies of the *Phas*^{T} allele were similar in C_0 and C_3. There was a highly significant linear trend in the frequencies of the *Phas*^{C} allele, while the changes in frequency of the *Phas*^{T} allele appeared to be due to random drift.

The frequencies of the *lec*$^-$, *Arc1*$^+$ and *phas*$^-$ alleles shifted in a manner consistent with theoretical expectations. The *lec*$^-$ allele, which has a positive effect on percentage phaseolin, increased markedly with each selection cycle. There was an increase in allele frequency of 199% from C_0 to C_3, and deviations and linear and quadratic effects were all significant. The frequencies of the *Arc1*$^+$ and *phas*$^-$ alleles, both of which have strong

Table 1. Frequencies and standard errors for six seed protein alleles from three cycles of selection for increased percentage phaseolin

Cycle	Seed protein allele					
	Phas^S	*Phas*^{C}	*Phas*^{T}	*lec*$^-$	*Arc1*$^+$	*phas*$^-$
C_0	0.574 0.018	0.147 0.013	0.147 0.013	0.276 0.016	0.147 0.013	0.129 0.013
C_1	0.637 0.028	0.153 0.022	0.170 0.020	0.349 0.026	0.045 0.009	0.038 0.006
C_2	0.559 0.047	0.374 0.043	0.068 0.019	0.678 0.043	0.00	0.00
C_3	0.461 0.046	0.385 0.041	0.153 0.027	0.826 0.027	0.00	0.00

Table 2. Chi-square tests for deviations from random drift and for linear and quadratic trends in gene frequencies for six seed protein alleles

Source	Seed protein allele					
	Phas^S	*Phas*^{C}	*Phas*^{T}	*lec*$^-$	*Arc1*$^+$	*phas*$^-$
Deviations	NS	*	NS	**	*	**
Linear	NS	**	NS	**	**	**
Quadratic	NS	NS	NS	*	**	**

*,** Significant at the 0.05 and 0.01 probability level, respectively
NS = Not significant

negative effects on percentage phaseolin, decreased rapidly and were eliminated from the population in the second cycle. Deviations and linear, and quadratic effects also were significant, indicating that the changes were too large to be due to random drift alone.

It was expected that this recurrent selection procedure would produce a reduction in the frequencies of *Phas*^{S} and *phas*$^-$ alleles and an increase in the frequencies of *Phas*^{C} and *Phas*^{T} alleles. Genotypes with *S*-type phaseolin often produce less phaseolin than do those with *C*- or *T*-type phaseolin (Hartana 1986). However, two of the parental lines used in this population (2-4-1 and 6-30) had *S*-type phaseolin but also gene(s) that enhanced phaseolin production. If these gene(s) were linked to the phaseolin locus, it would explain the initial rise in the frequency of the *Phas*^{S} allele. A break up of this linkage would account for the subsequent decrease in frequency.

There could be several explanations for the lack of a directional change in the frequency of the *Phas*^{T} allele. The frequencies of *Phas*^{S}, *Phas*^{C}, *Phas*^{T}, and *phas*$^-$ alleles are not independent, since they are at the same locus and only two alleles can be present in any diploid individual. Therefore, if the frequency of the *Phas*^{C} allele increases, and the frequencies of the *Phas*^{S} and *phas*$^-$ alleles do not decrease by at least an equal amount, then the frequency of the *Phas*^{T} allele must decrease, as it did in C_2. Some of the *Phas*^{T} alleles could also have been lost when parents were chosen to generate the new cycle population (i.e., random drift). This would also increase the frequency of other alleles at the same locus. Another possibility is that the *Phas*^{C} allele has a greater selective advantage than the *Phas*^{T} allele. It may be that in future cycles the frequency of the *Phas*^{S} allele would continue to decrease and the *Phas*^{C} and *Phas*^{T} alleles would increase as expected.

Proportion of variation explained by the protein alleles

The proportion of the phenotypic variation in percentage phaseolin that was explained by each of the markers changed with each cycle of selection. In the parental lines, where the alleles were all homozygous, *lec*$^-$, *Arc1*$^+$ and *phas*$^-$ accounted for large proportions of the variation (Table 3). Sixty-six percent of the varation in percentage phaseolin was explained by a model including *phas*$^-$ alone, and 98.6% of the phenotypic variation was accounted for by a model including all the alleles (Table 4). From C_1 to C_3, the magnitude of the R^2 values decreased greatly. In C_3 none of the alleles accounted for more than 0.5% of the variation and only 0.93% of the phenotypic variation was explained by all the alleles combined.

The low R^2 values reflect the fact that there were families with the same or nearly the same frequency of a particular allele, but with different amount of phaseolin. This indicates that effects associated with the alleles (or at least *Phas*^{S}, *Phas*^{C}, *Phas*^{T}, and *lec*$^-$) are not due solely to the alleles themselves, but may be due to other loci that are linked to the protein alleles. The change in magnitude of the R^2 values my be due to a breakup of these linkages. Another possibility, which does not necessarily assume any kind of linkage, is that effects of the protein alleles may become hidden as the frequency of other favorable alleles (for which we have no markers) increases with each cycle of selection. Also, as the alleles become fixed, genetic variance decreases and the R^2 decreases. If alleles are fixed within the population, the R^2 only reflects environmental variation.

Other studies have presented R^2 values for F_2 populations, which would be most similar genetically to C_1 in this experiment. Edwards et al. (1987) found that for 25 yield-related traits in maize, R^2 values ranged from 0.25% to 16.3% among 20 individual isozyme marker loci. Nienhuis et al. (1987) reported a range of R^2 values

Table 3. Percentage of phenotypic variation for percentage phaseolin explained by each of the seed protein markers individually for the original parental lines and the parents of the C_1, C_2, and C_3 generations

Marker	Parental lines	Parents of cycle ($R^2 \times 100$)		
		C_1	C_2	C_3
Phas^S	7.68	13.54***	0.48	0.19
Phas^C	5.05	1.55	0.71	0.46
Phas^T	3.90	7.13**	0.04	0.07
lec$^-$	18.62**	9.13**	4.12*	0.50
Arc1$^+$	17.75*	2.45	0.0	0.0
phas$^-$	66.28***	13.87***	0.0	0.0

*, **, *** Significant at the 0.10, 0.05 and 0.01 probability levels, respectively

Table 4. Percentage of phenotypic variation for percentage phaseolin explained by a model including all seed protein markers (*Phas*^{S}, *Phas*^{C}, *Phas*^{T}, *lec*$^-$, *Arc*$^+$, *and phas*$^-$) for the original parental lines and the parents of the C_1, C_2, and C_3 generations

Cycle	$R^2 \times 100$
Parents	98.62**
C_1	25.41*
C_2	5.15
C_3	0.93

*, ** Significant at the 0.05 and 0.01 level, respectively

Table 5. Percentage of each parent in the selected families over three cycles of selection for increased percentage phaseolin

Parent	Marker	Cycle			
		0	1	2	3
2-4-1	*Phas*^{S}	13.2	21.0	19.8	20.0
6-30	*Phas*^{S}	14.7	17.0	15.6	14.8
PP11-37	*Phas*^{C}	14.7	14.5	19.8	18.5
809598-3	*Phas*^{T}	14.7	17.0	12.5	13.5
L12-56	*Phas*^{S} and *lec*$^-$	14.7	19.7	21.4	21.1
SARC1-7	*Phas*^{S} and *Arc1*$^+$	14.7	8.0	8.9	9.6
BMC 3522	*phas*$^-$ and *lec*$^-$	13.2	2.6	2.1	2.3

from 5 to 17% for six RFLP markers associated with insect resistance in tomato. Those findings are consistent with the results presented here.

Contribution of parental lines to each cycle

The contribution of each parent in the base population and in the three cycles of selection was determined from the pedigrees of the selected families that composed each cycle. It was interesting that in spite of the fact that two of the alleles dropped out of the population in the second cycle, all seven parents were represented in the pedigree of plants in all cycles (Tables 5). The contribution of SARC1-7 and BMC 3522 decreased after C_0 but they were not eliminated from the population. One would have expected BMC 3522, which produces no detectable phaseolin, to have dropped out rather quickly; however, besides the *phas*$^-$ allele BMC 3522 also carries the *lec*$^-$ allele, which has a positive effect on percentage phaseolin. The percentage of each parent remained fairly stable after C_1. The major contributors were 2-4-1, L12-56, and PP11-37 with 20.0, 21.1, and 18.5%, respectively.

S_1 family recurrent selection was effective in shifting the frequency of seed protein alleles, however it does have some disadvantages. The probability that favorable alleles will be lost is higher for S_1 family selection than for other types of recurrent selection, especially if the alleles are present at low frequency (Choo and Kannenburg 1979). This very nearly occurred in this population for the *Phas*^{T} allele. Another disadvantage is that alleles become fixed in the population sooner than for other recurrent selection methods (e.g., half-sib selection), which rapidly reduces the genetic variance. This may partially explain the rapid increase in the frequency of the *lec*$^-$ allele and the decrease in frequency of the *Arc1*$^+$ and *phas*$^-$ alleles.

It is apparent from these results that the genetic control of phaseolin concentration is complex. As shown in Table 6, S_1 families with similar percentages of phaseolin can have very different allele frequencies. Obviously, many different combinations of the alleles can result in

Table 6. Frequencies of seed protein alleles in some families selected from cycle 2

Family no.	Seed protein allele						Percentage phaseolin
	Phas^S	*Phas*^{C}	*Phas*^{T}	*lec*$^-$	*Arc1*$^+$	*phas*$^-$	
1	0.283	0.242	0.475	0.417	0	0	14.8
2	0.592	0.408	0	0.350	0	0	14.8
3	0.642	0.358	0	1.000	0	0	14.6
6	0.417	0	0.583	0.976	0	0	14.3
7	0	0.525	0.475	0.750	0	0	14.2
8	1.000	0	0	1.000	0	0	14.2
10	0.092	0.558	0.350	0.433	0	0	14.1

high phaseolin accumulation. It is also likely that other favorable alleles are segregating within this population. Although the seed protein alleles examined in this study had large effects on percentage phaseolin in the early generations, other alleles also must be important. The results indicate that the effects associated with some of the alleles (e.g., $Phas^S$) may not be the effects of a single locus, but may be due to two or several loci acting together. It would be interesting to isolate the gene(s) that enhance phaseolin production in the parental line 2-4-1, since it comprises 20% of the pedigrees in the population and thus has a major effect.

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