

ADDITIONAL EVIDENCE BEARING ON THE PLEIOTROPIC ACTION OF AM-1 AND AM-2

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Past reports (1, 2) pointed to a direct (pleiotropic) relationship between the genes am-1 or am-2 and one or the other of two associated seed disorders. The association was interpreted as pleiotropy rather than linkage, even though some am-1 or am-2 plants and lines do not express the seed disorder. The explanation offered was that the seed disorder fails to manifest in the presence of one or more genes that counteract one aspect (the seed disorder) but not the other aspect (flower color inhibition) of gene action. The gene b was suggested as playing a role in the scheme.

To investigate the matter further, crosses were made between a wild-type line (A, B, Am-1, Am-2, F/Fs) and several A/A lines with white flowers (due to am-1 or am-2 or both) but not showing a seed disorder. The results of one of these crosses are considered here (Table 1). It is evident from the segregation pattern: (i) that the white-flowered parent carried only one flower color inhibitor gene; (ii) that the gene in question was am-2 (distinguished by the nature of the seed disorder found in F2); (iii) that since b segregated in F2, b was present but masked in the white-flowered parent; and (iv) that some but not all the white-flowered F2 plants showed a seed disorder. Further, the relative number of white-flowered segregants with and without seed disorder could be explained by assuming the segregation of a recessive gene pair that blocked the pleiotropic action of am-2. The b gene was a likely candidate in that role.

Table 1. Number of plants with indicated phenotypes in the F₂ of a cross between a line with wild-type flowers and a line with white flowers (but A/A), both having normal seeds.

Population	Observed/Expected number of <u>F₂</u> plants		<u>F₂</u> phenotype	
	Obs.	Exp. (9:3:3:1)	Flower color	Seed
B280-819-829	196	186.2	Wild-type	Normal
	63	62.1	Pink	Normal
	56	62.1	White	sd-2 ^{1/}
	16	20.7	White	Normal
	331	331.1		

^{1/} sd-2 indicates a seed disorder of a type typically associated with the presence of am-2 and marked by self-colored olive green seeds, many of which show various degrees of degeneration.

The F2 results also implied that if b/b serves as the block, then some of the white-flowered segregants with the seed disorder (presumably B/-) should, when progeny tested, segregate for plants without the seed disorder because some am-2/am-2 segregants were bound to be B/b (am-2 and b apparently are not linked). This prediction was tested by growing 20 F3 progenies from selected white-flowered F2 plants. Two pink-flowered (b/b Am-2/-) F2 plants were also progeny tested.

Each F3 progeny consisted of 12 plants. This is not enough to identify all potentially heterozygous (B/b) entries but sufficient to detect heterozygosity in most progenies and therefore to test the validity of the hypothesis. The results of the FT analysis are given in Table 2. Of 12 plants that showed

seed disorder in F₂, ten segregated for plants with normal seed (i.e. without seed disorder) in F₃, giving a combined ratio of 87 segregants with and 32 segregants without seed disorder ($\chi^2[3:1]=0.227$). In three of the F₃ progenies, all 12 plants exhibited the seed disorder and therefore possibly were homozygous B/B. Seven of the F₂ plants tested were without the seed disorder in the field. All 12 plants in each of the corresponding F₃ progenies were again without the seed disorder. Presumably, therefore, all seven progenies were b/b. The two b/b Am-2/- F₂ plants produced F₃ progenies that were entirely free of the seed disorder, including three am-2 plants that segregated in one of the b entries.

Table 2. Progeny test results of 21 F₂ plants derived from the cross A Am-2 B x A am-2 b. Each F₃ progeny consisted of 12 seeds from an individual F₂ plant.

F ₂ Phenotype		F ₃ Phenotype			Presumed F ₂ genotype
Flower	Seed	Flower color	Seed		
			Normal	sd-2	
White	Normal	White	12	0	<u>A am-2 B/B</u>
"	"	"	12	0	
"	"	"	12	0	
"	"	"	12	0	
"	"	"	12	0	
"	sd-2	"	3	9	<u>A am-2 B/b</u>
"	"	"	5	7	
"	"	"	2	10	
"	"	"	6	6	
"	"	"	2	10	
"	"	"	2	9	
"	"	"	2	10	
"	"	"	2	10	
"	"	"	3	9	
"	"	"	5	7	
"	"	"	0	12	<u>A am-2 b/b</u>
"	"	"	0	12	
"	"	"	0	12	
Pink	Normal	All Pink	8 ^{1/}	0	<u>A Am-2/am-2 b/b</u>
"	"	9 Pink:3 White	12	0	

1/ Only 8 seeds progeny tested

In a different but related experiment, it was shown that, after selfing a line heterozygous for A (A/a) but homozygous for am-1, all the A/- segregants showed the seed disorder which is associated with am-1 and all the a/a segregants had normal seed. This same behavior was noted in three successive generations, F₄, F₅, and F₆. Thus a₋ is epistatic to am-1 both in terms of flower color and seed disorder.

It seems therefore that A and B are required for the seed disorders associated with am-1 and am-2 to become phenotypically manifest. Once A and B are present, then the specific type of seed disorder that is expressed depends on whether am-1 or am-2 or both are present (2). A seemingly complex pattern of dominance and interaction (epistasis) emerges from all this, a situation perhaps made clearer by the following scheme:

Gene Locus ^{1/}				Phenotype of	
A	B	Am-1	Am-2	Flower	Seed
+	+	+	+	Wild-type	Normal
+	+	+	-	White	sd-2
+	+	-	+	White	sd-1
+	+	-	-	White	sd-2
+	-	+	+	Pink	Normal
+	-	+	-	White	Normal
+	-	-	+	White	Normal
+	-	-	-	White	Normal
-	+	+	+	White	Normal
-	+	+	-		
-	+	-	+		
-	+	-	-		
-	-	+	+		
-	-	+	-		
-	-	-	+		
-	-	-	-		

^{1/} (+) implies dominant (-) implies homozygous recessive

Many of my earlier, unreported, observations which initially defied explanation have now begun to make sense in the light of this proposed scheme. I have reason to believe that still another gene may interact with am-1 and am-2 to override the seed disorder phase of gene action but the analysis is not yet complete.

1. Marx, G. A. PNL 7:28-29. 1975.
2. Marx, G. A. PNL 10:38-40. 1978.