Table S1. Molecular lesions and phenotypic strength of amp1 alleles

A. Molecular lesions of amp1 alleles

Mutant	Origin	Ecotype	Position*	Sequence change	Mutagen† EMS	
amp1-1	Chaudhury et al., 1993	Col	2225 bp	G to A (Stop)		
amp1-8	This work	Le <i>r</i>	976 bp	C to T (Stop)	EMS	
amp1-9	This work	Ler	1434 bp	G to A (3' acceptor splice site)	EMS	
amp1-10	This work	Col	1112 bp	SALK_021406	T-DNA	
amp1-11	This work	Col	1129 bp	SALK_038402	T-DNA	
amp1-12	This work	Col	1133 bp	SALK_044086	T-DNA	
amp1-13	This work	Col	2461 bp	SALK_022988	T-DNA	

^{*}The origin of mutation was mapped by PCR. The full-length AMP1 genomic sequence is 2964 bp in length and comprises 10 exons.

B. Phenotypic strength of amp1 alleles

Genotype	n	Monocot %	Fused [‡] %	Dicot %	Tricot %	Tetracot %	Pentacot %	Collar§ %	Twin¶%
Col-0	169			100					
amp1-1	172		1.7	76.1	21.5	0.6			
amp1-12	167	0.6	6.0	50.3	37.7	3.0		1.8	0.6
amp1-13	154	0.6	10.4	46.1	39.6	1.9		0.6	0.6
amp1-10	130	0.7	14.6	44.6	36.1	0.7		3.1	
amp1-11	147	2.7	10.2	41.5	44.2	0.7			0.7
Ler	167			100					
amp1-8	189		3.1	60.8	34.9	1.1			
amp1-9	213	0.5	5.1	48.3	39.2	6.1		0.5	
pt	198	0.5	2.5	48.4	45.5	2.5	0.5	0.5	

The phenotypic spectrum of cotyledon number indicates which *amp1* alleles are considered weak and strong, increasing in severity as the number of cotyledons increase. Seedlings were classified at 5 DAG. The *amp1-1* allele displays milder defects, which is consistent with the residual *AMP1* gene activity in this allele. *pt*, Primordia timing.

[†]EMS, ethylmethanesulfonate; T-DNA, insertion of T-DNA.

^{*}Fused cotyledons have one or more fused cotyledons.

[§]Collar seedlings form a continuous ring of tissue surrounding the SAM.

Twin seedlings develop more than one embryo from a single seed coat.