Table S3. χ^2 calculations for double-mutant seeds

Hypotheses		Phenotypic classes [WT]	[*dme*]	[*atlig1*]	[*atlig1*/*dme*]	χ^2	Degree of freedom	*P*
AtLIG1 and *DME* are in independent pathways and *atlig1; dme* seeds show both phenotypes	Obs	34	75	21	0			
	Exp (%)	26	13.7	13.7	46.6			
	Exp (*n*=131)	34	18	18	61			
	(Obs-Exp)^2/Exp	0	180.5	0.888888889	61	242.3888889	4–1=3	*P*<0.001
The *atlig1* and *dme* mutations are synthetic lethal and *atlig1; dme* seeds abort	Obs	34	75	21	0			
	Exp (%)	26						
	Exp (*n*=131)	34	18	18	61			
	(Obs-Exp)^2/Exp	0	180.5	0.888888889	61	242.3888889	4–1=3	*P*<0.001
atlig1 is epistatic over *dme* and *atlig1; dme* seeds have an *atlig1* phenotype	Obs	34	75	21	N.E.			
	Exp (%)	26	60.3	13.7	–			
	Exp (*n*=131)	34	18	79	–			
	(Obs-Exp)^2/Exp	0	180.5	41.12658228	–	221.6265823	3–1=2	*P*<0.001
dme is epistatic over *atlig1* and *atlig1; dme* seeds have a *dme* phenotype	Obs	34	75	21	N.E.			
	Exp (%)	26	13.7	60.3	–			
	Exp (*n*=131)	34	79	18	–			
	(Obs-Exp)^2/Exp	0	0.202531646	0.888888889	–	1.091420534	3–1=2	*P*>0.5
Both mutations complement each other and *atlig1; dme* seeds have a WT phenotype	Obs	34	75	21	N.E.			
	Exp (%)	72.6	13.7	13.7	–			
	Exp (*n*=131)	95	18	18	–			
	(Obs-Exp)^2/Exp	39.16842105	180.5	0.888888889	–	220.5573099	3–1=2	*P*<0.001

(WT) male will produce 25% WT seeds, 25% *dme* seeds and 25% *atlig1; dme* seeds. Owing to the incomplete penetrance of each mutation observed in the single-mutant control crosses (see Table 2), the proportion of *atlig1* and *dme* seeds showing a phenotype will be 22.25% (0.25 × 0.89) and 17.5% (0.25 × 0.7), respectively. Thus, the proportion of expected WT seeds is 35.25% [0.25 + (0.25 – 0.2225) + (0.25 – 0.175)]. However, 35.25% of 523 is 184 seeds, and we obtained 218 WT seeds. The 34 extra seeds (218 – 184) are most likely to be double-mutant *atlig1; dme* seeds that show a WT phenotype due to the incomplete penetrance of each mutation. However, it is not possible to predict the degree of penetrance for each mutation in this double-mutant background as the penetrance is expected to vary depending on the genetic background. Thus, we calculated that out of 131 genetically *atlig1; dme* seeds (25% of 523), 34 are phenotypically WT, which enables us to calculate that the penetrance of both mutations amounts to 74% [(131 – 34)/131]. Based on this result, we estimated that the penetrance of each mutation in the double-mutant background was 86% (sqrt of 0.74). Therefore, out of 131 genetically double-mutant seeds, we expect that 34 will have a WT phenotype, 18 will have a *dme* phenotype, 18 will have an *atlig1* phenotype, and 61 will show a phenotype resulting from the interaction of both mutations. The obtained data only fit a model in which *dme* is epistatic over *atlig1* (χ^2=1.09, *P*>0.5), which indicates that *AtLIG1* acts downstream of *DME*, most likely by repairing the DNA breaks created by DME in the central cell.

Obs, observed.

Exp, expected.

N.E., none expected.

A female *atlig1-3/+; dme-4/+* crossed with a wild-type