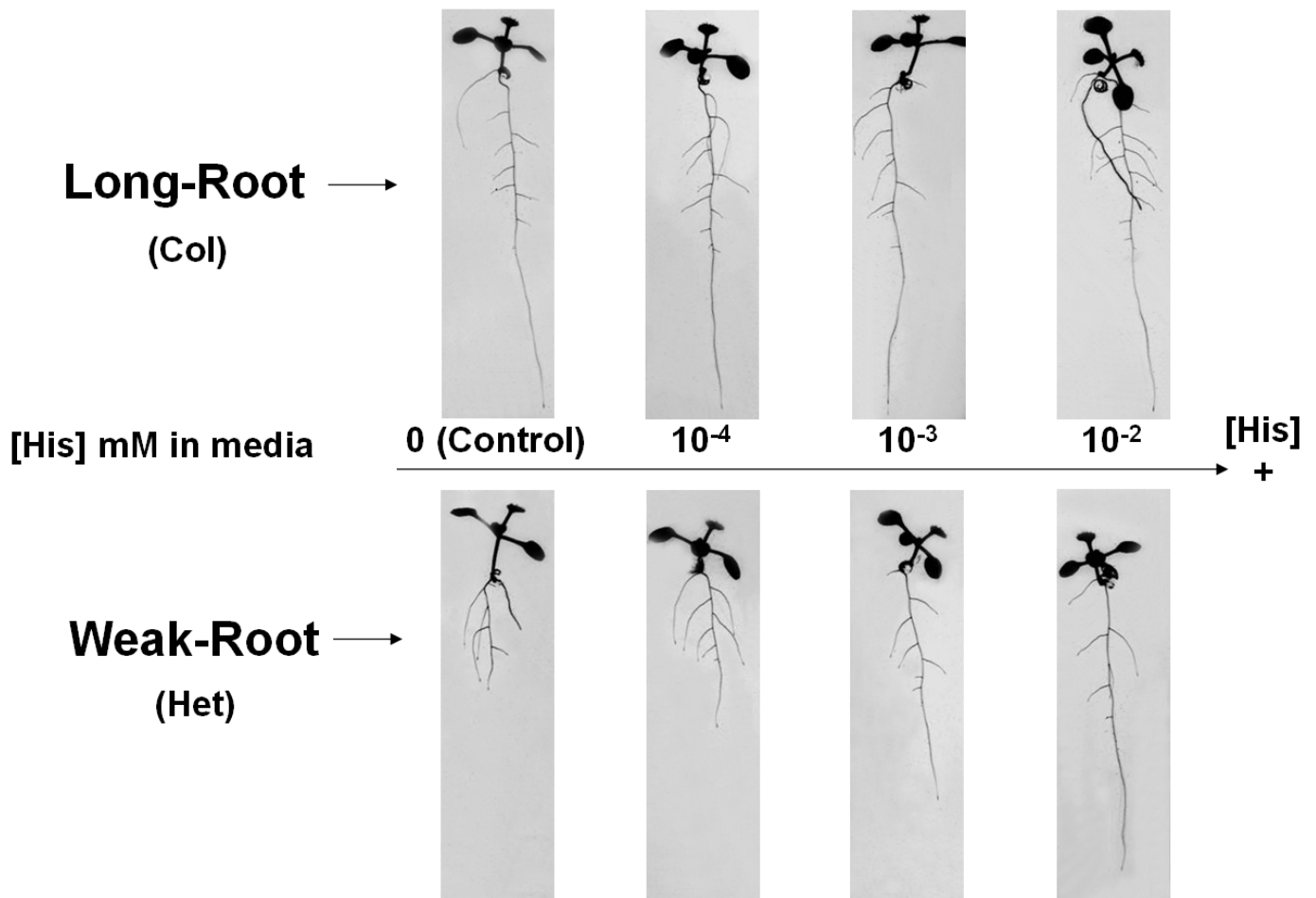


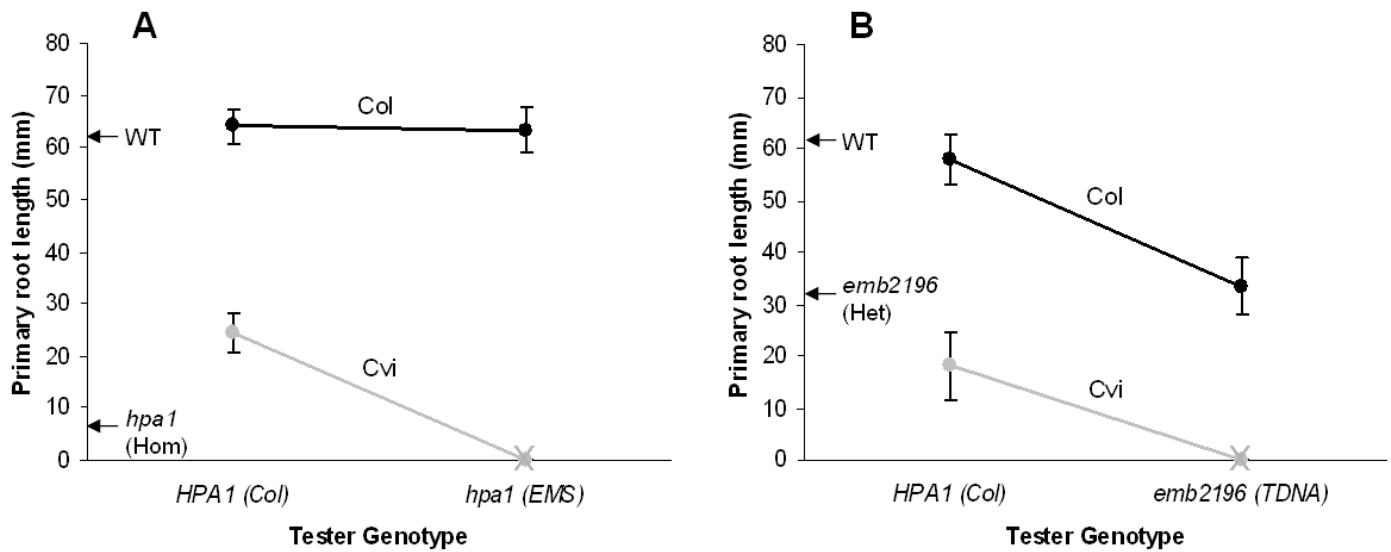
**Figure 1 (Bikard et al., Science, 2009)**

Genetic mechanism underlying LD1 interaction and incompatibility. All combinations of alleles at LD1.5 and its interactor LD1.1 result in phenotypically normal plants, except for the combination Col at LD1.1/Heterozygous (Het) at LD1.5, which shows a reduced primary root length (*weak-root* phenotype), and another combination (Col at LD1.1/Cvi at LD1.5), which shows embryo lethality at an early stage in the silique (seeds under development containing lethal embryos are indicated by arrows). Fine-mapping identified a duplicate gene for which Col and Cvi show reciprocal gene loss explaining the interaction and incompatibility.



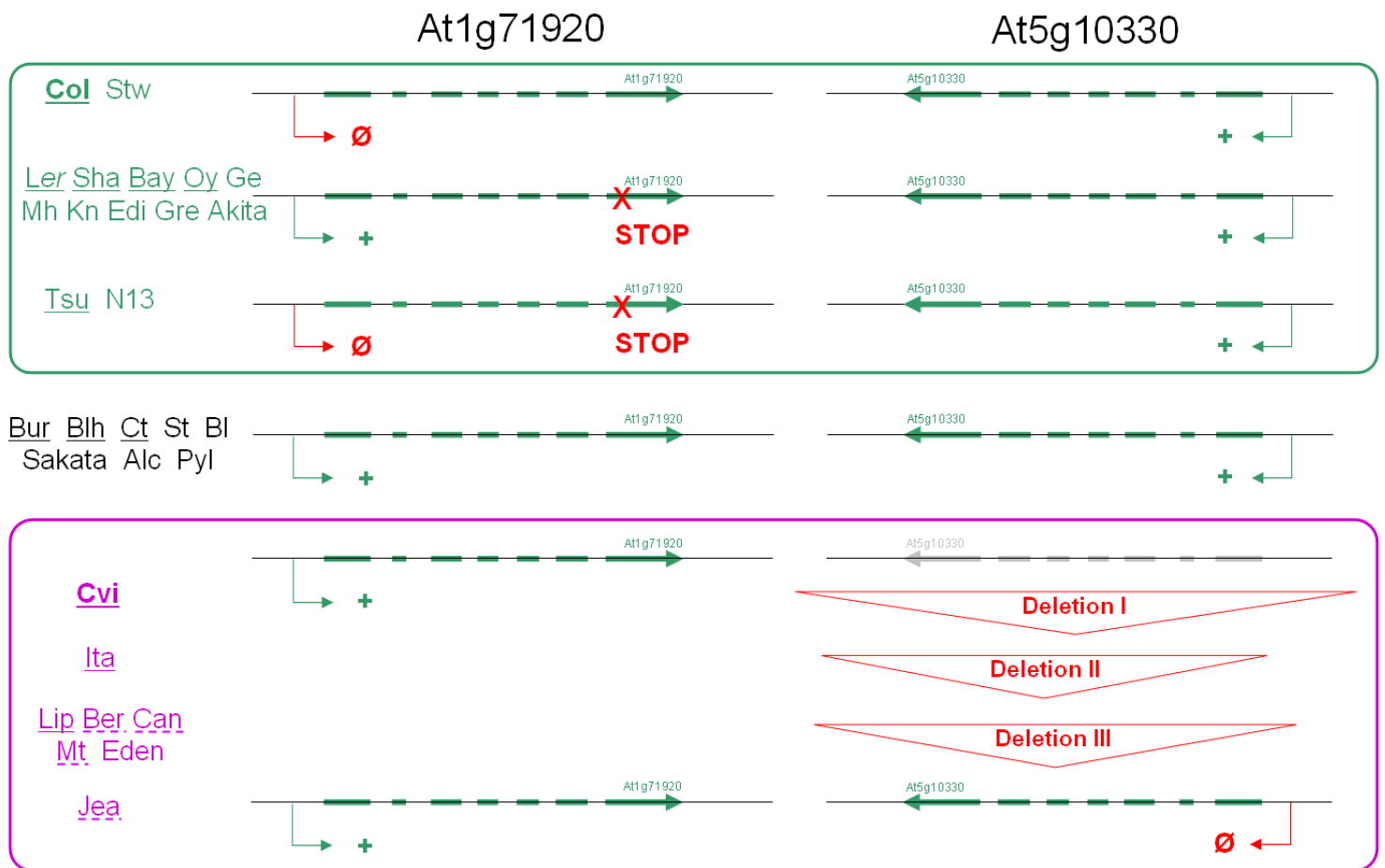
**Figure 2 (Bikard et al., Science, 2009)**

The weak-root phenotype is quantitatively complemented by exogenous His. Typical phenotypes of descendants from a plant segregating at the LD1.5 locus when grown on media supplemented with different histidine concentrations. Plants were identified on the basis of their genotype at the LD1.5 locus. The complementation of the expected weak-root phenotype was complete when supplied with  $10^{-2}$  mM His.



**Figure 3 (Bikard et al., Science, 2009)**

Allelic complementation of LD1 interaction. Quantitative complementation tests were performed by combining different alleles at LD1 loci in F1 backgrounds. The relative complementation of either (A) an EMS mutant allele (*hpa1*) or (B) a T-DNA insertion mutant allele (*emb2196*) at At5g10330 by a Col or Cvi allele at LD1.5 was measured through both the length of the primary root (shown in mm) and embryo lethality. Individuals with a phenotype depicted on the x axis (null) underwent seed abortion. All plants are Col at LD1.1. Arrows along the y axis represent the typical root length of control genotypes [WT indicates wild-type Col plants; *hpa1* (Hom), homozygous *hpa1* mutant plants; and *emb2196* (Het), heterozygous individuals for the *emb2196* mutation]. Each data point represents the mean  $\pm$  standard error of about 40 plants. The LD1.5 allele  $\times$  HPA1 genotype interaction term as tested by analysis of variance is significant in crosses to *hpa1* ( $P < 0.0001$ ) and crosses to *emb2196* ( $P < 0.05$ ).



**Figure 4 (Bikard et al., Science, 2009)**

Divergent evolution of duplicate genes among *A. thaliana* accessions. Groups of accessions are presented according to At5g10330 and/or At1g71920 genotypes and transcript accumulation phenotypes. Accessions (underlined) from each group were crossed to Col and/or Cvi and tested for LD1 incompatibility and compatibility to confirm the loss of function of one or the other duplicate gene. Dash-underlined accessions show conditional incompatibility (table S1). The incompatible groups are circled: green-circled genotypes are incompatible with Cvi; purple-circled genotypes are incompatible with Col. Genotypes not circled are fully compatible with both Col and Cvi.