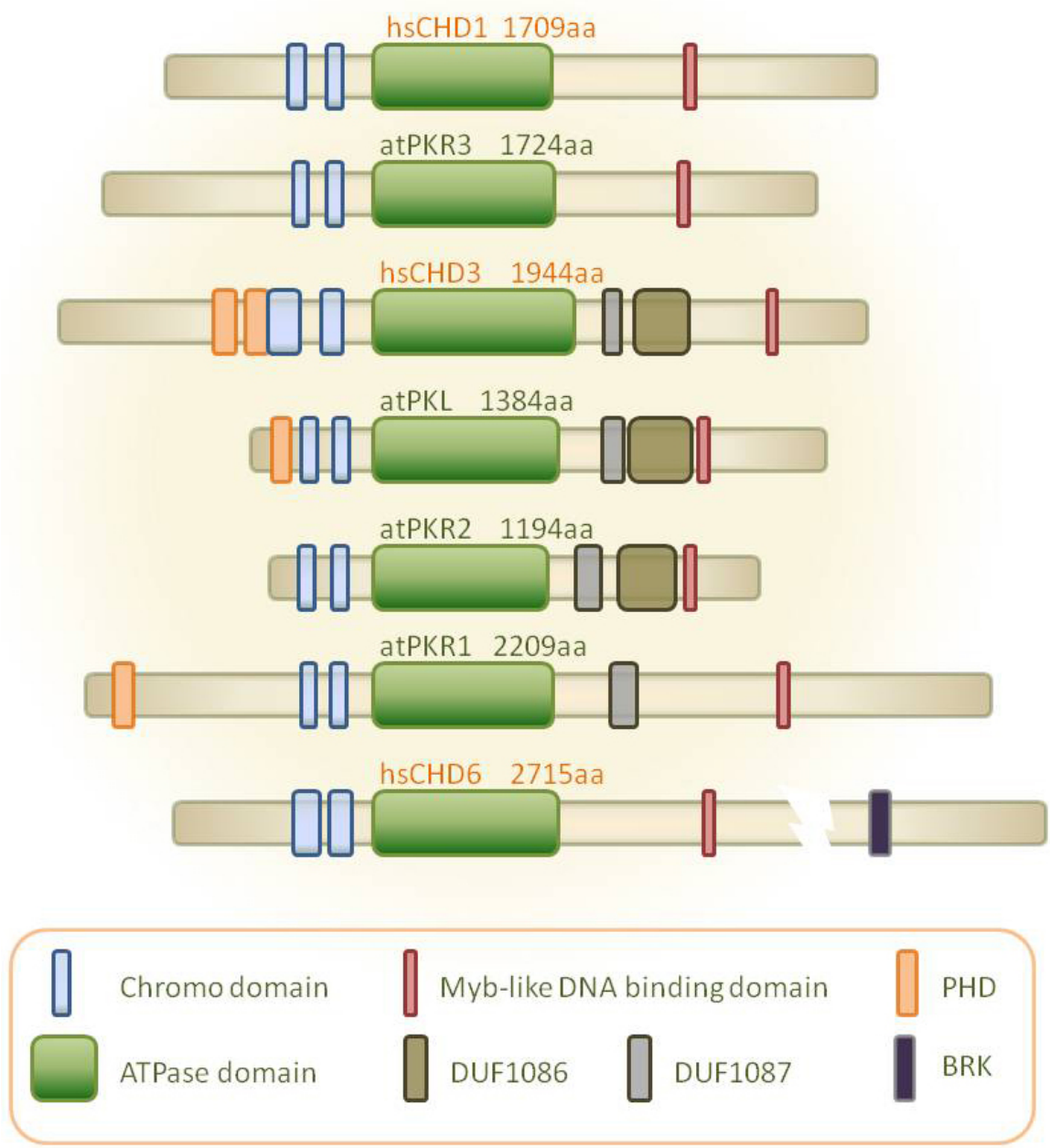


Figure S1. Related CHD proteins from animals and plants have similar organization of domains. CHD proteins from *H. sapiens* (hs) and *A. thaliana* (at) are drawn to scale with known or predicted domains included.

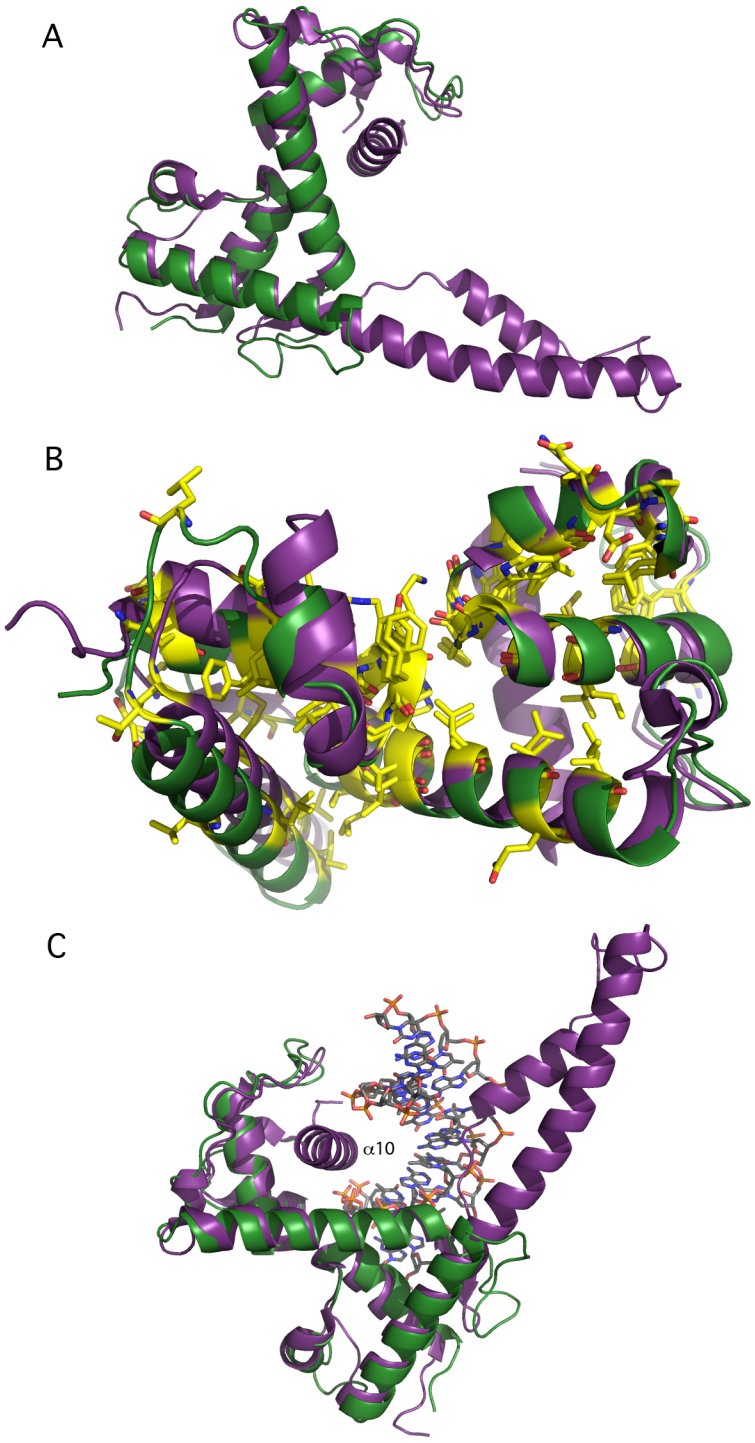


Figure S2. The predicted structure of the *A. thaliana* (at) PKL DNA-binding domain is similar to the DNA-binding domain of CHD1 from *S. cerevisiae* (sc). scCHD1 is shown in purple whereas atPKL is shown in green. (A) Overlap of atPKL and scCHD1. (B) Overlap of atPKL and scCHD1 with orientation of conserved amino acid residues depicted in yellow. (C) Overlap of atPKL and scCHD1 with characterized position of DNA relative to scCHD1 included.

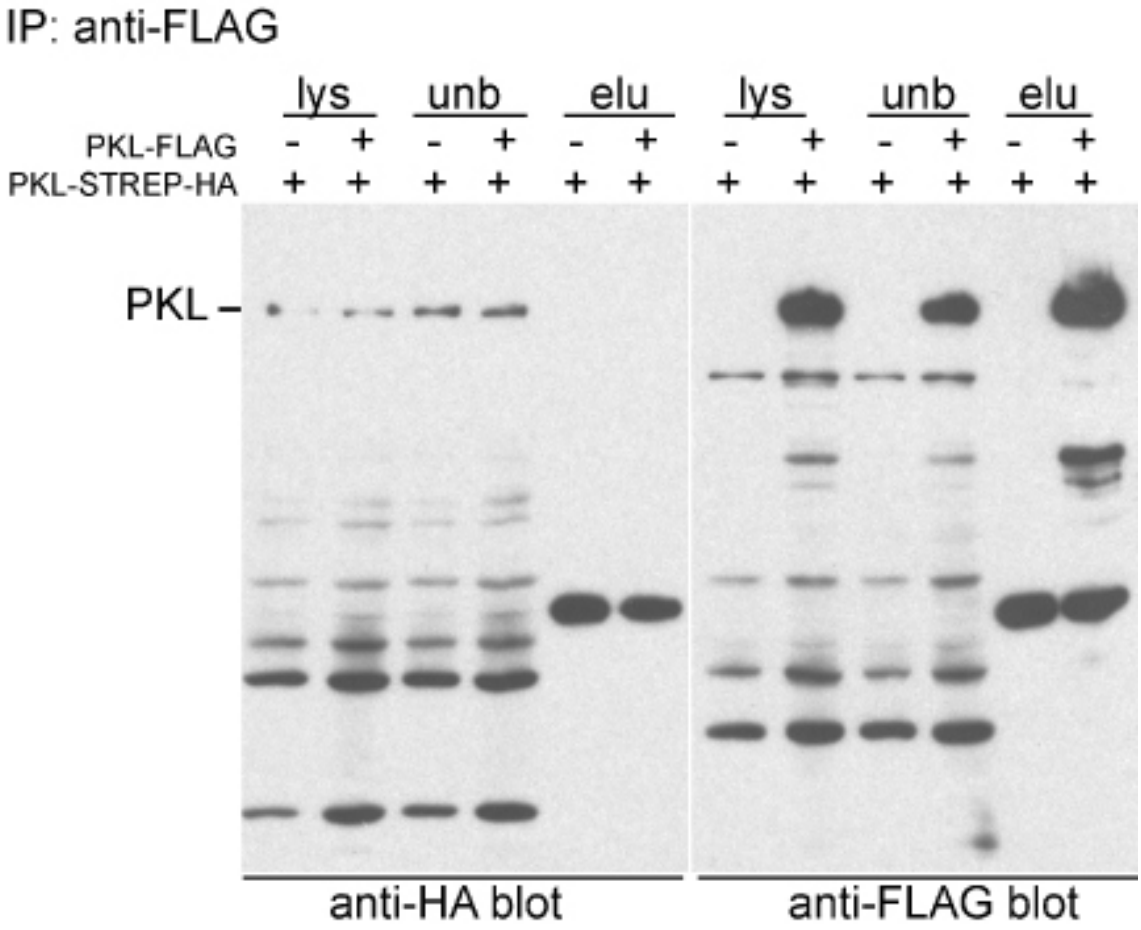


Figure S3. Co-immunoprecipitation analysis does not reveal self-association of PKL protein. Anti-FLAG antibodies were used to immunoprecipitate PKL-FLAG from plants that expressed PKL-FLAG or from plants that expressed both PKL-FLAG and PKL-STREP-HA. Both the *PKL-FLAG* transgene and the *PKL-STREP-HA* transgene are under the control of endogenous *PKL* regulatory elements and either transgene is sufficient to rescue all mutant phenotypes associated with loss of *PKL*. Western analysis using anti-HA antibodies (anti-HA blot) or anti-FLAG antibodies (anti-FLAG blot) was used to examine levels of PKL-FLAG and PKL-STREP-HA proteins respectively in total lysate (lys), the fraction of sample that does not co-immunoprecipitate with PKL (unb), and the fraction of sample that does co-immunoprecipitate with PKL-FLAG (elu). No PKL-STREP-HA is detected in the fraction that co-immunoprecipitates with PKL-FLAG.