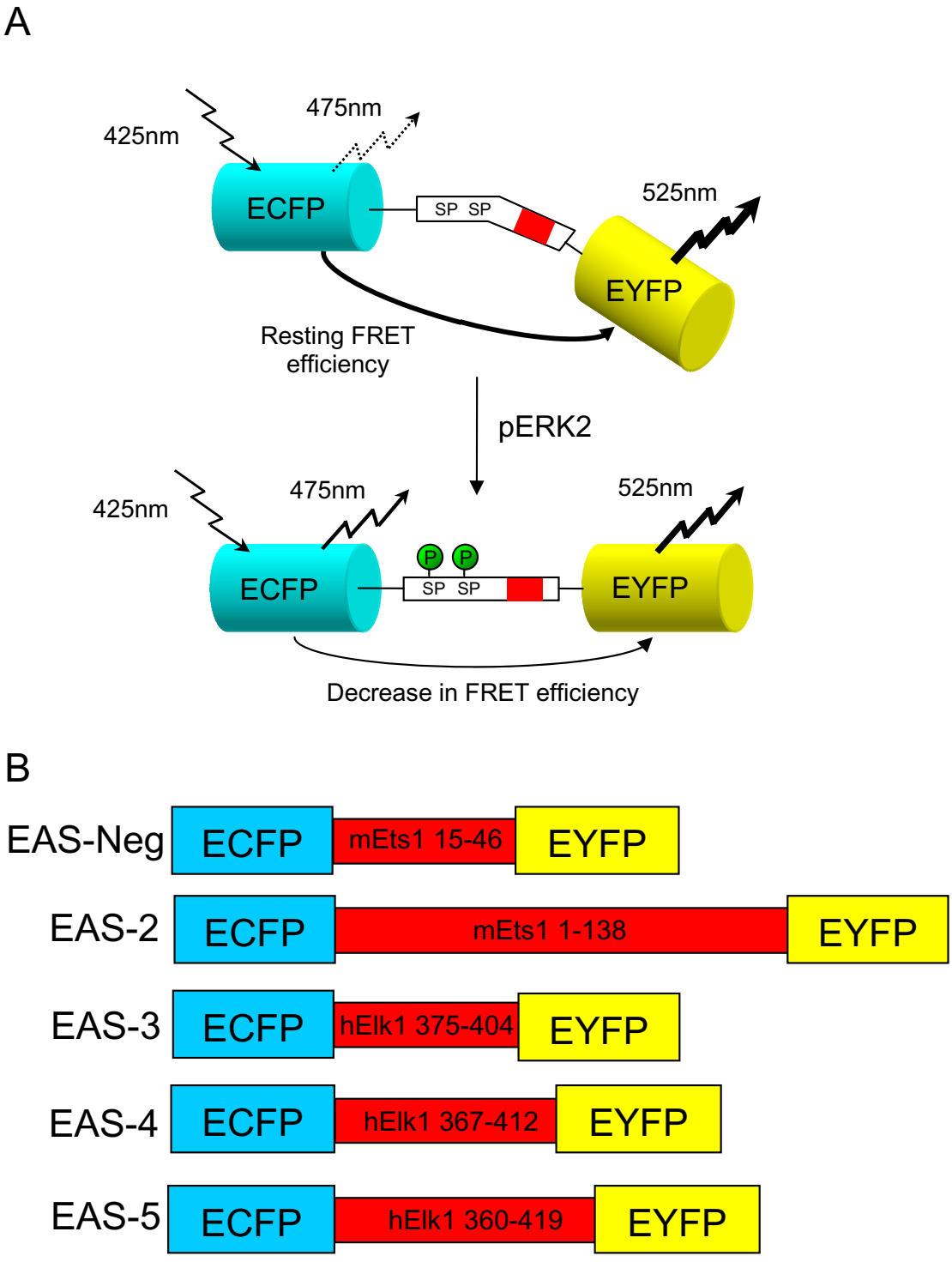


**Figure 1**

**A model for EAS activation and construct design**. (A) Our inferred model indicates that upon pERK2 phosphorylation a conformational change in the linker peptide of EAS decreases the efficiency of FRET. The red area in the linker peptide indicates the relative position of either D-domain (EAS-2) or DEF domain (EAS-3, -4, -5) and the SP motifs denote the consensus phosphorylation sites. (B) The gene constructs include EAS-Neg and EAS-2, which are derived from mouse Ets1. EAS 3–5 contain peptide linkers derived from human Elk1. The composition of each linker peptide is indicated by the primary sequence positions derived from Ets1 and Elk1, respectively.