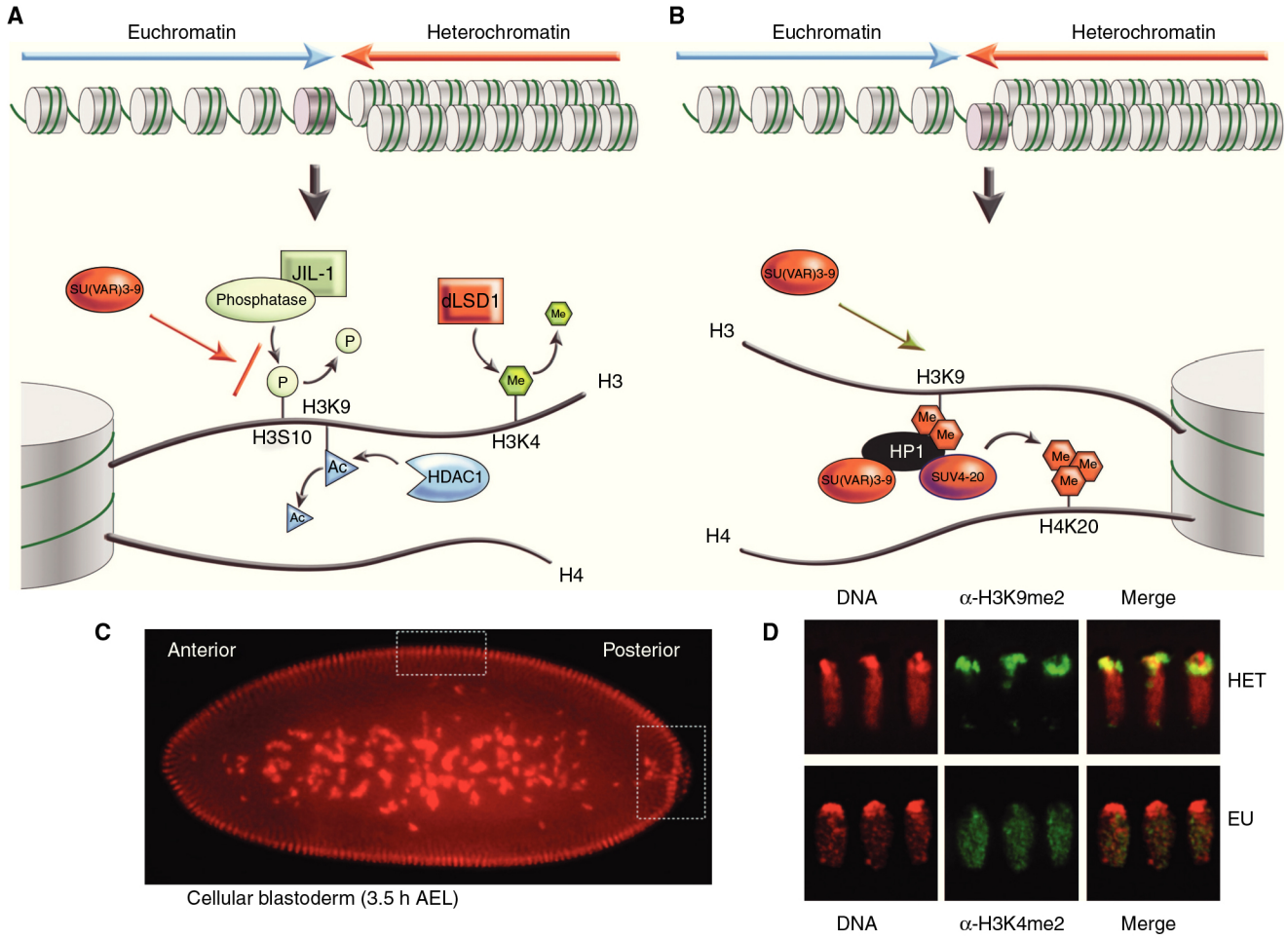

Figure 6. The transition from a euchromatic state to a heterochromatic state requires a series of changes in histone modification. (*A*) Active genes are marked by H3K4me2/3; if present, this mark must be removed by LSD1. H3K9 is normally acetylated in euchromatin; this mark must be removed by a histone deacetylase, HDAC1. Phosphorylation of H3S10 can interfere with methylation of H3K9; dephosphorylation appears to involve a phosphatase targeted by interaction with the carboxyl terminus of the JIL1 kinase. These transitions set the stage for acquisition of the modifications associated with silencing, shown in *B*, including methylation of H3K9 by SU(VAR)3-9, binding of HP1a, and subsequent methylation of H4K20 by SUV4-20, an enzyme potentially recruited by HP1a. (*C*) Differentiation of euchromatin and heterochromatin is initiated in early embryogenesis around cell cycle 10 and is completed when cellular blastoderm (*top* box) and primordial germline cells (*right-hand* box) are formed. (*D*) Blastoderm nuclei show an apicobasal polarity (Rabl conformation). Heterochromatin (H3K9me2 staining) is established at the apical site, whereas euchromatin (H3K4me2 staining) is organized toward the basal site. (Immunofluorescent images provided by Sandy Mietzsch.)