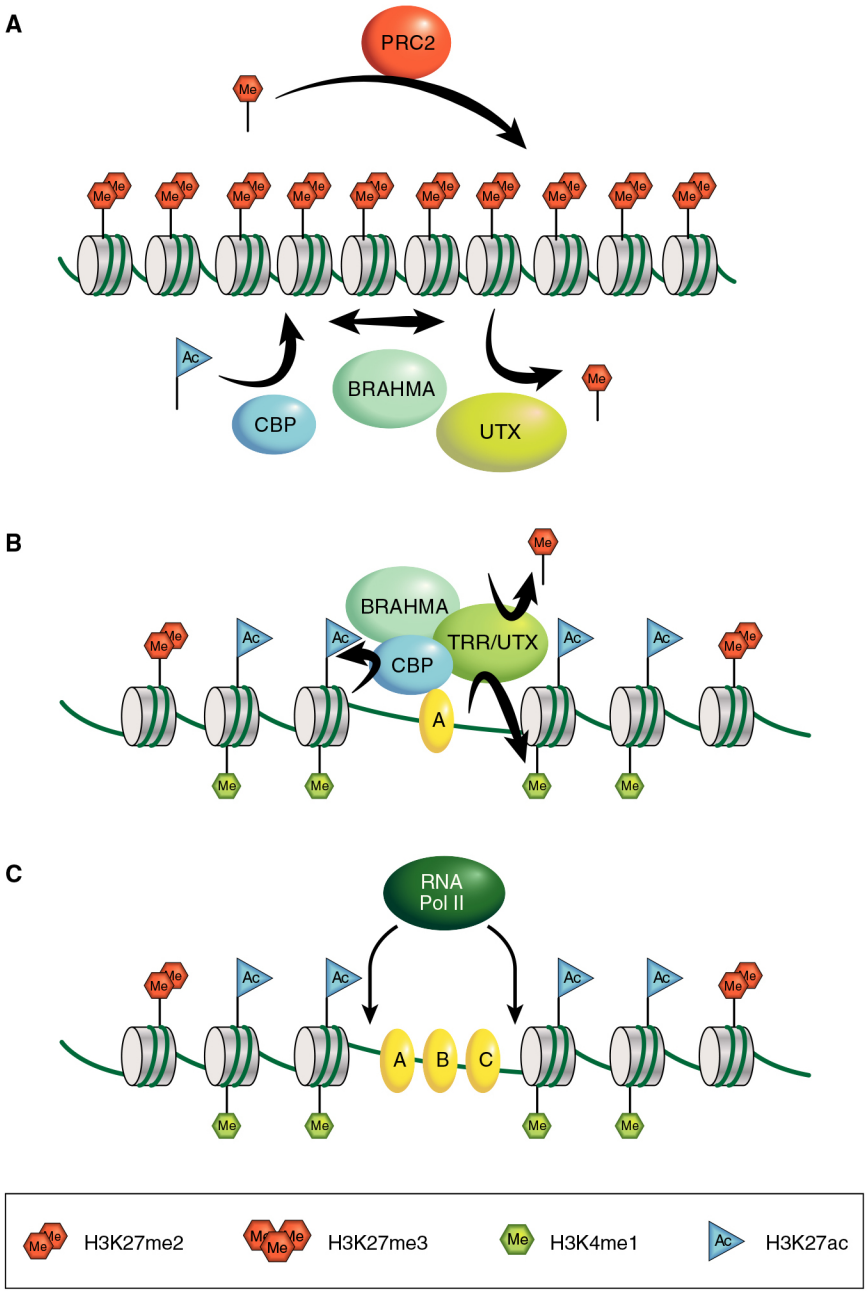


Figure 2. A model for the control of DNA accessibility in chromatin. (*A*) The model proposes that antagonistic roaming activities transiently interact with genomic chromatin: one, caused by PRC2, deposits the H3K27me2 mark. Another removes this methylation mark and remodels nucleosomes, allowing transient access to the DNA sequence. These activities are attributed to UTX, CBP, and BRAHMA. (*B*) A DNA-binding factor A binds to its cognate binding motif in the DNA, transiently made accessible, and recruits stable binding of CBP together with a remodeling activity (BRAHMA) and the TRR/MLL3,4 complex containing UTX. These activities remove H3K27 methylation, depositing instead the H3K27ac and H3K4me1 marks. (*C*) The remodeling activity provides stable access to the DNA, leading to the binding of additional factors B and C to an enhancer region (or other regulatory element on the DNA). The region of DNA made accessible can also be opportunistically targeted by RNA polymerase, which may produce short transcripts from both DNA strands.