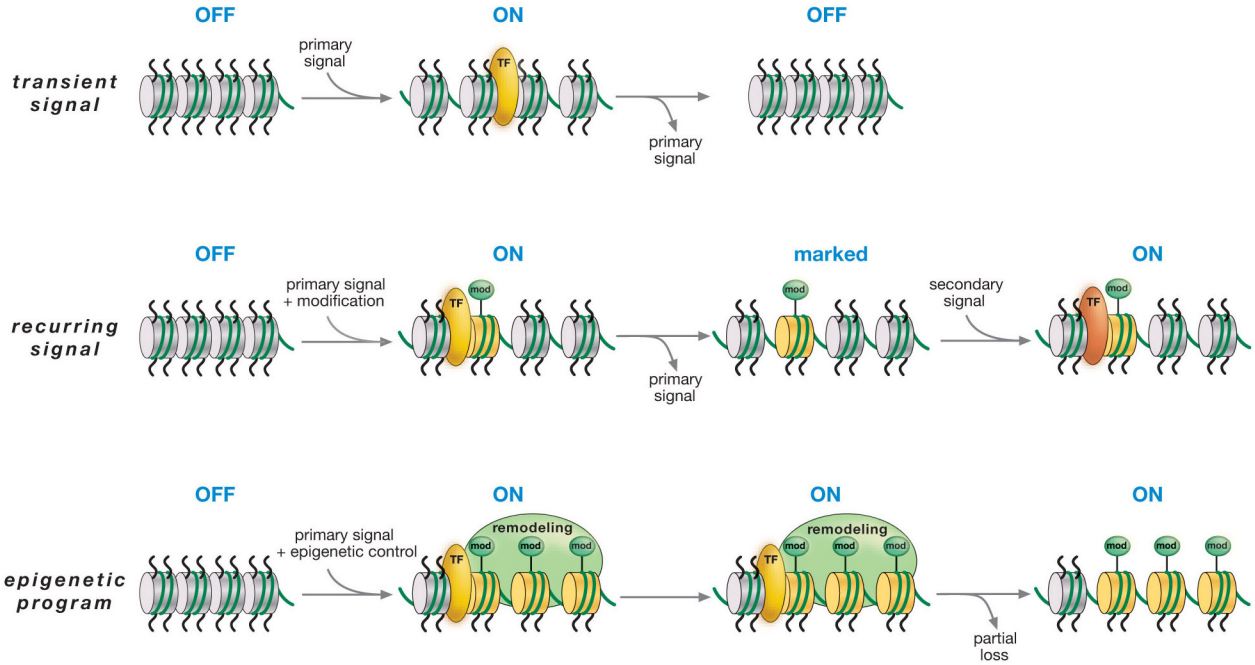

Figure 20. Epigenetic Potentiation of a Primary Signal (Memory/Inheritance)

Classic genetics predicts that gene expression is dependent on the availability and binding of the appropriate panel of transcription factors (TF). Removal of such factors (i.e., a primary signal) results in the loss of gene expression, and thus constitutes a transient activating signal (*top*). Chromatin structure contributes to gene expression, where some conformations are repressive and others active. The activation of a locus may therefore occur through a primary signal and result in the downstream change in chromatin structure, involving active covalent histone marks (mod) and the replacement of core histones with variants (e.g., H3.3). Through cell division, this chromatin structure may only be reestablished in the presence of an activating signal (denoted "recurring signal"). Epigenetic memory results in the maintenance of a chromatin state through cell division, even in the absence of the primary activating signal. Such a memory system is not absolute, but involves multiple levels of epigenetic regulation for remodeling chromatin structure. The dynamic nature of chromatin means that although a chromatin state may be mitotically stable, it is nonetheless prone to change, hence affecting the longevity of epigenetic memory.