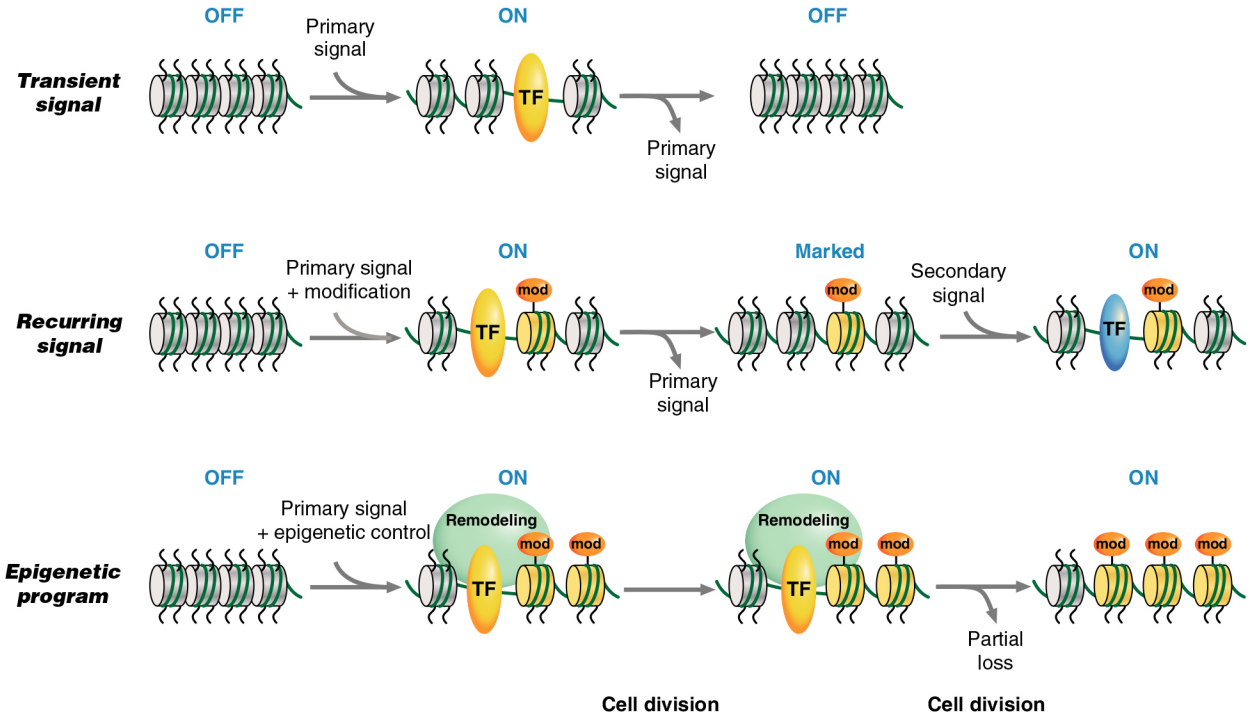

Figure 37. Epigenetic transmission of a primary signal. Classical genetics predicts that gene expression is dependent on the availability and binding of the appropriate panel of TF. Removal of such factors (i.e., a primary signal) results in the loss of gene expression and, thus, constitutes a transient activating signal (*top*). Chromatin structure contributes to gene expression, in which 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 activating 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 or a recurring secondary 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 stable during mitosis, it is nonetheless prone to change, hence, affecting the longevity of epigenetic memory.