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A Propagation of an epigenetic mark



B Replication-dependent propagation of an epigenetic mark

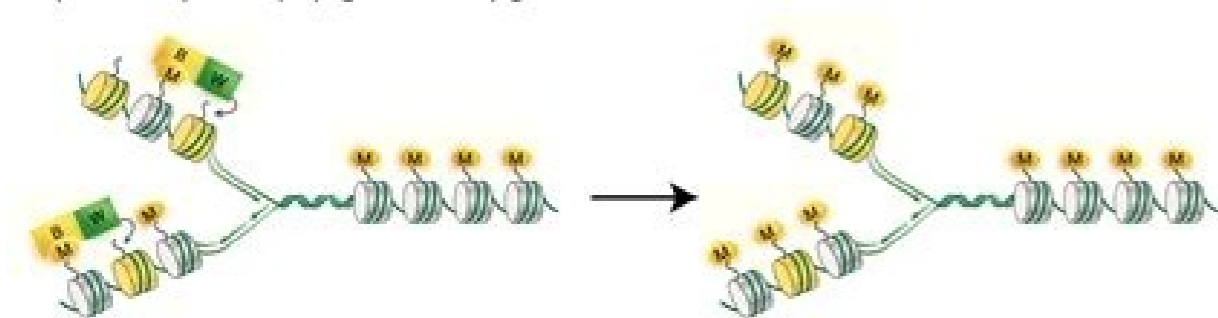


Figure 1. Propagation of epigenetic marks. (A) A general mechanism for propagating a histone modification such as H3K9 methylation typically found in heterochromatic regions. The modified histone tail (M) interacts with a protein binder (B) that has a binding site specific for that modification. B also has a specific interaction site with an enzyme “writer” (W) that carries out the same histone modification on an adjacent nucleosome (gray cylinder). Spreading of the histone mark will continue until the modifying machinery reaches a boundary element, delineating the boundary between heterochromatin and euchromatin. (B) A general mechanism for maintaining a histone modification during replication. Newly deposited nucleosomes (yellow), which may incorporate histone variants, are interspersed with parental nucleosomes (shaded in gray) following DNA replication. The modified histone tail (M) on the parental nucleosome interacts with a protein binder (B). As in A, B interacts with a “writer” (W), which catalyzes a histone modification on the histone tail from an adjacent daughter nucleosome.

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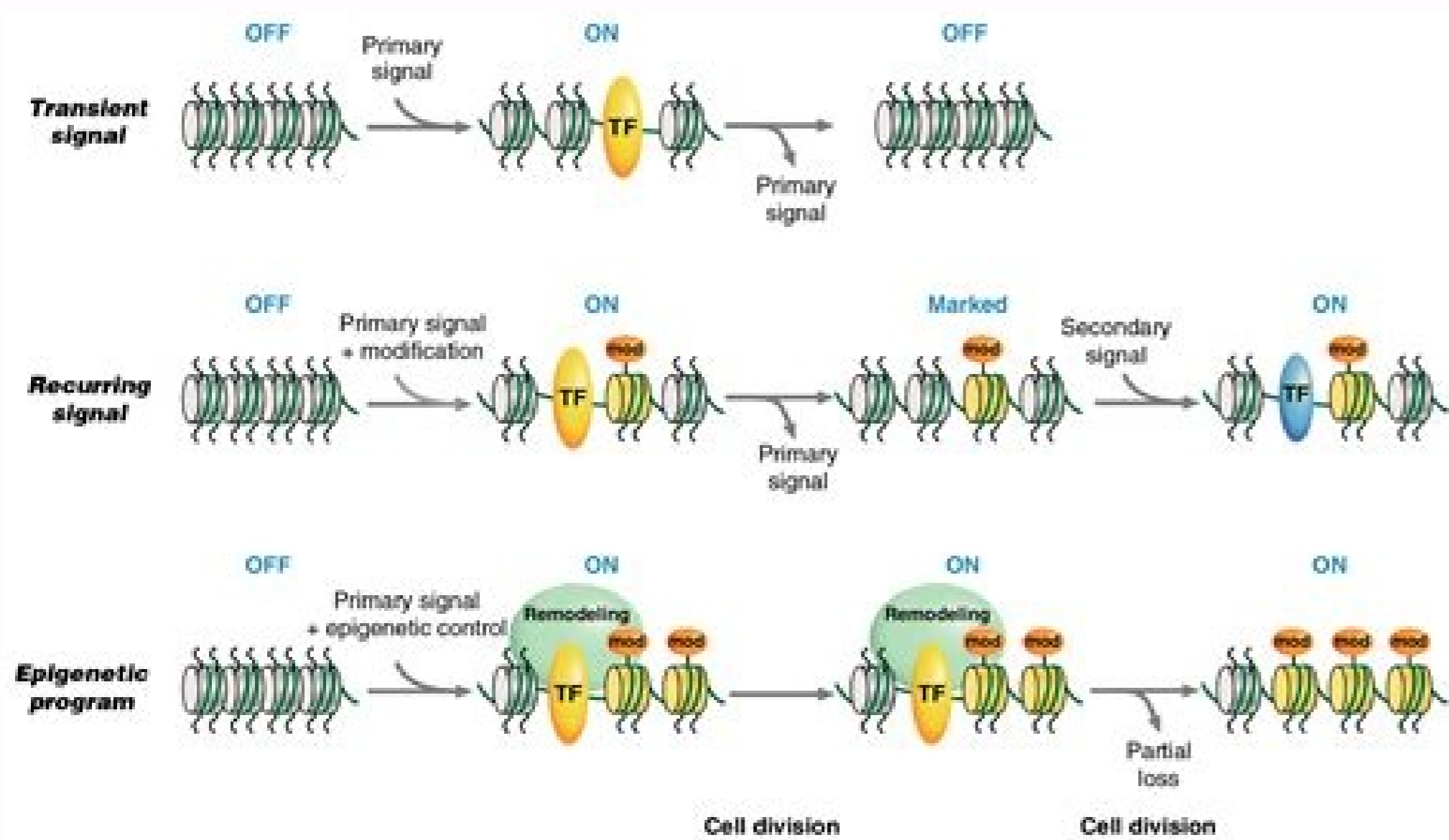
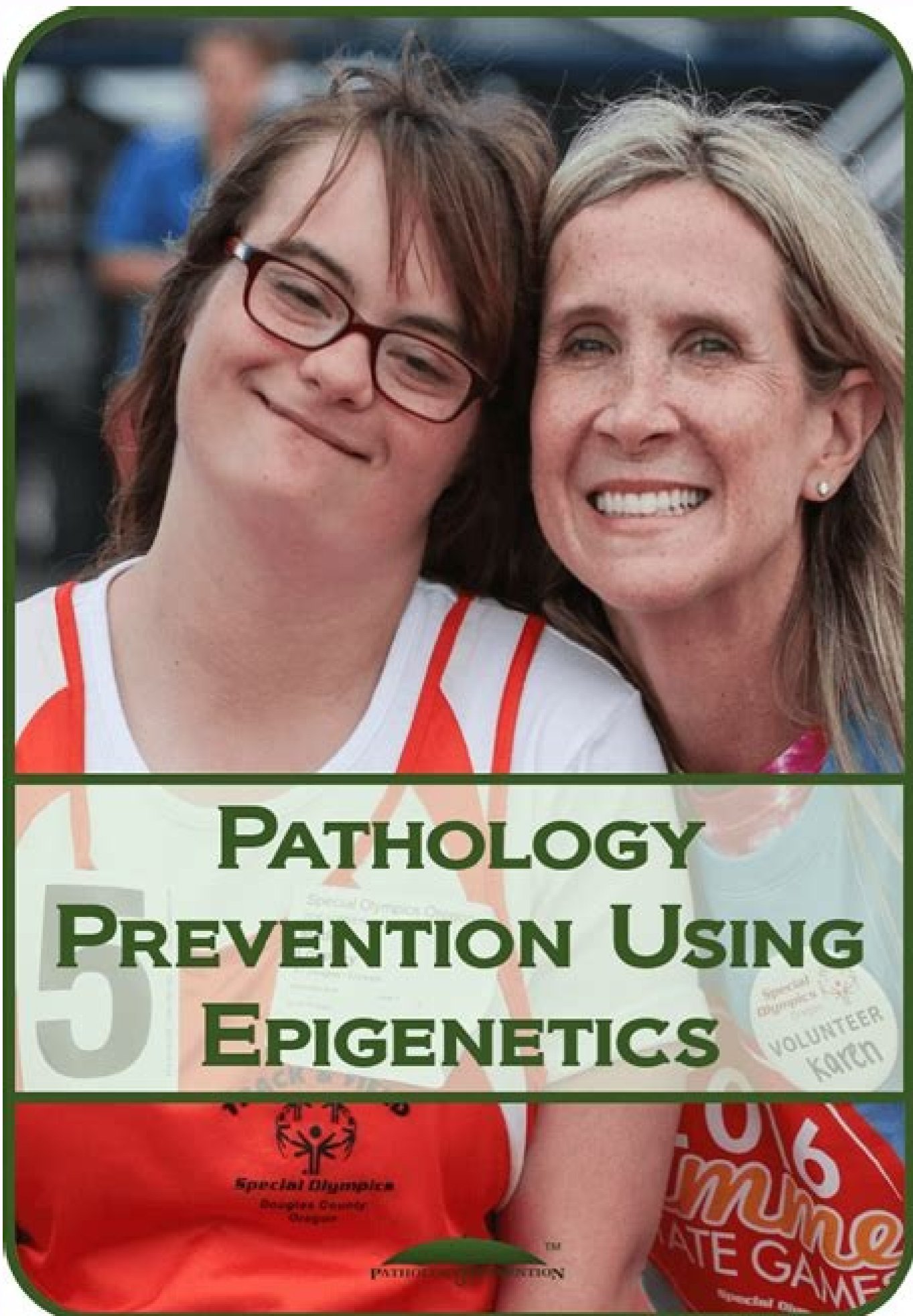


Figure 37. Epigenetic transmission of a primary signal. Classical genetics predicts that gene expression is dependent on the availability and binding of the appropriate panel of TF. Removal of such factors (i.e., a primary signal) results in the loss of gene expression and, thus, constitutes a transient 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.

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Contributions to Science Work and Society
 * The use of the word "epigenetics" in this article is not intended to imply any causal relationship between the two.
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