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## AN EASY WAY TO ELIMINATE CAUSES OF AUTOIMMUNE AND ALLERGIC DISEASES

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ur recent analytical study suggests that breast and ovarian cancers possibly have similar epigenetic origin. Interestingly, combination therapy, including epigenetic drugs, was effective against breast and ovarian cancers causing more than additive growth inhibition in various types of breast and ovarian cancer cells. Traditional therapies do not kill cancer progenitor cells and drug-resistant cancer cells, causing cancer relapse. Other studies have shown that combination therapy with epigenetic drugs reduced cancer relapse, sensitized drug resistant cancer cells, and killed cancer stem cells. We rationalize that initiation of cancer progenitor cell formation from predisposed cells requires an epigenetic switch. Further progression-development of cancer involves mutations. This hypothesis differs from current paradigm of carcinogenesis based on mutations. CpG residue methylation in the upstream regions of genes is one of the epigenetic regulations involved in silencing of tumor suppressor genes in cancer cells. In addition, histone modifications, such as H3K4me, and H3K27me3, and other histone modifications regulate gene expression in concert with alterations in DNA methylation. Our system biology analysis revealed that DNA methyl transferase1 (DNMT1), the enzyme which maintains CpG residue methylation is allosterically activated in cancer cells. We hypothesized that the combination of CpG DNA methylation and histone modifications constitutes an epigenetic switch. It is possible that histone modification and CpG methylation work in a concert (alteration in epigenetic switch) to regulate differential gene expression in carcinogenesis.