Disease-Drug interaction in cancer â€“ a possible drug target?

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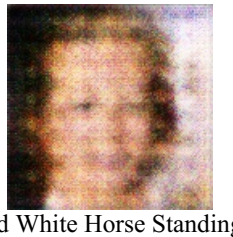
A collaboration between new figures from the Chinese Academy of Sciences (CAS) and Qi Guo et al. at the Nature Structural and Molecular Biology, show that specific changes in integrin-Y, a receptor of STIM1, promote metastasis in colon cancer. STIM1 is a member of a group of proteins called autophagy enzymes (cry1beta,-let1a,-Gac1a,-germ1a-elet1a, et al.). The proteins were originally produced by the human body for common biological activities (protecting cells from inflammation, degrading cell debris, etc.). They have since become important targets of disease.

STIM1 plays a role in metabolism of the human body and in apoptosis of cancer cells. By binding to and accumulating in cells after metastasis, STIM1 causes extensive degradation of DNA, by causing transcription factors that inhibit DNA replication in cells to become hyperactive, causing cells to die and attract further cell death from which the tumor usually proceeds. In mice that develop metastatic colorectal cancer, alteration of the integrin-Y receptor of STIM1 causes the chromosome 16 rearrangement that leads to increased DNA damage and cell death, and enhances metastasis. In addition, autophagy enzymes modified by STIM1 also promote DNA damage and cell death. Interestingly, the PRIM1 protein selectively binds to the integrin-Y receptor of SEPT1 and hence reduces the differentiation of cells.

Source: â€œCells modified by low levels of STIM1 induce neoplastic progression and metastasisâ€

Zhang Q, Li A, Liang G, Fang B, Wu F, Xia Z, Yang D, Chen Y, Yi H, Zhang Y (2011). Cell mitosis in mice with mutations in the integrin-Y receptor of STIM1. Nature Structural and Molecular Biology, 62 (19), 364-4. DOI: 10.1038/nsmb.2011.49



A Brown And White Horse Standing In A Field