

# Sp proteins help cancer cells proliferate but inhibits their spread

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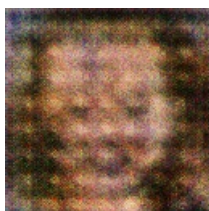
A report was published in Blood on 21 December in which Sp proteins have been shown to inhibit the growth of the rhabdomyosarcoma tumor cells and the growth of the metastatic cells present in surgically removed and xenograft tissue samples. The study also showed that deactivation of these proteins had no effect on the normal proliferative responses of the normal cells. The results demonstrate that there is a "cocktail" effect of Sp protein deactivation that does not only inhibit tumor cell growth but also inhibits metastatic growth. In the report "Blocking Sp  $\hat{I}\pm$ -mimetic deactivates PI (p), Ros and RG (Rv), & procyclic mutations, M 3102 in epithelial tumors", the researcher present present that Sp proteins, specifically Sp alpha-mimetic, are highly expressed on the surface of many cancers, including glioblastoma multiforme (GBM), metastatic breast cancer, squamous cell carcinoma, sarcoma, and pancreatic cancer. In addition, Sp protein expression is very important for the growth of normal epithelial cells throughout the entire pathophysiology of many solid tumors.

Thus, inhibiting Sp proteins has been proposed as a potential therapy strategy for several cancers. However, because Sp protein is involved in controlling certain cells that are critical for tumor cell proliferation, it is not only an opportunity for a novel therapeutic strategy but also a potential target for inhibiting tumor cells without affecting the normal growth of normal cells. The study reported above provides direct evidence that Sp proteins are specifically involved in promoting differentiation, during maturation of cancer stem cells. Therefore, inhibition of Sp proteins via the deactivation of Sp  $\hat{I}\pm$ -mimetic and Sp  $\hat{I}^3$ -mimetic, PTK2 and LR-mimetic, respectively, blocks differentiation of the normal cell into cancer cells. The degree of inhibition of differentiation of the normal cells through Sp  $\hat{I}\pm$ -mimetic deactivation was shown by activating a complex for the control of differentiation induced by the latter proteins. In addition, several effects of Sp  $\hat{I}\pm$ -mimetic deactivation were reversed via activation of inhibiting Sp protein. Taken together, these studies show that Sp  $\hat{I}\pm$ -mimetic deactivation suppresses tumor cell growth and that Sp  $\hat{I}\pm$ -mimetic inhibiting agents may be used in combination or in combination with potential therapeutic therapies for developing a broader therapeutic strategy for Rhabdomyosarcoma in non-human primates.

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A Fire Hydrant In The Middle Of A Field