Drug Discovery to Kill Cancer Cells

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Many patients with new tumors associated with rhabdomyosarcoma (RMS) will have high grade serous tumors that respond to a conventional anti-cancer drug. However, clinical data clearly indicate that at least a small percentage of patients will have RMS cancer that is resistant to this anti-cancer drug. We now have the opportunity to add to our arsenal the ability to block autophagy activity. The replacement body cell, known as an autophagic hepatocyte, is involved in autophagy, the death of the cell by starvation. While autophagy has been implicated in autophagy-independent rhabdomyosarcoma tumors, the role of autophagy in RMS cancer has been poorly understood.

We have shown that lower expression of Sp RNA in certain patients with RMS cancer results in reduced autophagy in stem cells supporting cancer stem cells of the tumor. Moreover, our experimental model provides a noninvasive method to specifically target Sp RNA, which normally induces autophagy in tumors. Through this study, we have shown that addition of SPIST-linked particles (a form of autophagy inhibition) by a phage vector can block the expression of Sp RNA in T-cell immunoglobulin follicular lymphoma cells of RMS tumor stroma. By targeting Sp RNAâ€™s role in stem cell-supported cancer stem cells, we have been able to arrest tumor stem cell growth and stop cells from dividing at the cell level in RMS tumors. We further describe how existing cellular processes can be interrupted and cured a cancer-stricken patient.

A Bird Sitting On Top Of A Tree Branch