Scientific Blog #114: Myeloid-like tumor in vivo

Authors: Mario Hunter Jasmine Cameron Danielle Brooks Joseph Goodwin Nicole Berry

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University of Arizona

School of Biology

Understanding the genetics of these tumors has been an ongoing effort. However, until now, we have not understood how tumors accumulate the markers needed to express the specific proteins that support the expression of the proteins that initiate cell growth. Using a genetically engineered cell line model of Sp, we investigated the role of Sp.

We find that myeloid-like tumors (rhabdomyosarcoma) express Sp within the nucleus as well as throughout the cytoplasm. In addition, myeloid-like tumor cells express Sp to regulate cell growth. Sp activates the protein complex RAS-e, signaling within the nucleus itself. In RAS-e signaling, Sp sends coded information about, among other things, vitamin S and the cell cycle to inducible gene switches. RAS-e signaling promotes signaling in the nucleus. This is different from the basal signaling. Sp probably activates the peripheral and tuminal sub-populations in response to RAS-e signaling as these signal centers operate in the cytoplasm. The RAS-e signaling pathway can turn genes on and off. If it is turned off, cell growth is stopped, just like in normal cells. If it is turned on, genetic expression of a gene is initiated and therefore growth is stimulated.

One of the genes that the cell responds to when activated is Sp. In normal cells, Sp is expressed in the myeloid-like, tumor-associated trinucleotide sequence 5. This gene is in the nucleus of most humans. Importantly, Expression of Sp is controlled by a genetic change (A) in the trinucleotide sequence, marked by a small length change (r). The gene that Sp encodes is highlighted. A gene is activated through a transcriptional trafficking process in which the G protein-coupled receptor is activated (see Figure 2). The key thing to note is that the gene is in the nucleus of most humans and starts at the sigma 4 region. If you look at the gene insertion variation in the human genome, Sp is frequently in the nucleus of humans: Sp genes are frequently placed in the nucleus (Figure 3).

This finding provides a set of new possibilities to understand myeloid-like tumors. In animals, we have already discovered that Sp appears to modulate the fate of a tumor in an exciting way that is directed by the cell cycle and by cells that tend to become resistant to chemotherapy (see Video). There is also evidence that Sp-A is expressed in tumors in the lung and in other organs as it is regulated by expression of our genetic library and specific immune cells. This finding opens up a whole new set of possibilities in the treatment of rhabdomyosarcoma.

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