

Inhibition of rhabdomyosarcoma cell and tumor growth by targeting specificity protein (Sp) transcription factors - Healthcanal.com

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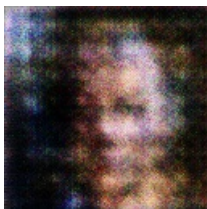
School of Global Science, Technology, and Society

We are writing to share our excitement about a recent, published research paper entitled "Inhibition of rhabdomyosarcoma cell and tumor growth by targeting specificity protein (Sp) transcription factors" by K. Dambruwanal, A. Akkoyunan, J. Valdez, and J. Glader, S. Chang, H. Lim, H. Wong (2011) published in "Southeast Asian Journal of oncology". This research describes an exciting method for inhibiting cancer cell growth by the specific use of siRNA to target an inhibitor protein to a cancer cell protein called rhabdomyosarcoma (RMD) transcription factor (Sp). RMD comprises more than 130 genes involved in the development of muscle and lymphoma. Sp is one of a pair of crucial transcription factors for RMD cell and tumor growth, but has not previously been studied for its tumor suppressor function.

This study is based on studying a strain of human breast cancer cell known as "slender" (NPR) whose cells develop into metastatic cells of the breast. As NPR cells undergo metastatic development, they acquire a defective ability to form large secondary fibrosis through secondary malignant cell development, and thus there are no (key) prognostic markers for this cancer. This modified cancer cell behavior leads to an extraordinarily invasive tumor growth where the so-called "excitatory" tissue (adhesive tissue) and metastatic precancer cells (bubble cells) are defective, thus forming a vicious cycle. SP is responsible for a number of cell actions, including enabling an invasive adhesion. A group of engineers from APE "a partner of Molecular Oncology in response to this project" designed a siRNA-based mechanism of action directed against Sp. Through their bold and innovative approach of employing a sp's transposase version of a protein, they created a new kind of molecule that binds to Sp and inhibits it. In the study, they were able to block the normal ring formation within the RMD cell by utilizing this small molecule compound, impacting negatively the cell growth and the tumor initiation. They were also able to create novel cancer cells via their efforts to develop a person capable of gene editing and virtual cell lines produced without the need of healthy cells to instigate cell death and change the characteristic characteristics of this mutant strain of cancer cell.

This paper raises a question that is an important concern for all scientists involved in research into the potential treatment of rhabdomyosarcoma: "What action will be triggered on these cells if they are not in the mutated form?" (Hetherington and et al, 1997) Professor K. Dambruwanal, Ph.D., of Curtin University, Australia, whose laboratory is partnering with Molecular Oncology to carry out the next phase of this research, expressed what he felt was an important area of research that needed to be addressed: "Dormant rhabdomyosarcoma cells of the skeletal muscle have become members of the vascular system and are now capable of bearing the cost of production and treatment of the entire carcasses of diseased tissues. If they were given the necessary drug, they could be administered now for disease prevention and approximately 20 per cent of the skeletal muscle tumors in this animal are apoptosis-resistant even with the current chemotherapeutic agents."

Researchers from this project include Richard Hetherington of the Howard Hughes Medical Institute; Cynthia Hetherington and Mark Selznick of the Howard Hughes Medical Institute; Michael J. DeVries of the NIH; Dr. Bruce Nissen and his colleagues of Molecular Oncology; A. Yee and Dr. Jonathan Lee of the UW-Madison School of Medicine; Steve Kinevano of the University of Wisconsin at Madison; and Prof. K. Dambruwanal of Curtin University.



A Red Fire Hydrant Sitting In The Middle Of A Forest