New way to put the brakes on cancer found

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While great strides have been achieved in cancer treatment, scientists are looking for the new targets and next generation of therapeutics to stop this second leading cause of death nationwide. A new platform for drug discovery has been developed through a collaborative effort linking chemists at NYU and pharmacologists at USC.

In a study appearing in *Proceedings of the National Academy of Sciences*, the research groups of Paramjit Arora, a professor in NYU's Department of Chemistry, and Bogdan Olenyuk from the USC School of Pharmacy have developed a synthetic molecule, "protein domain mimetic," which targets the interaction between two proteins, called transcription factor-coactivator complex at the point where intracellular signaling cascade converges resulting in an up-regulation of genes that promote tumor progression.

This approach presents a new frontier in cancer research and is different from the typical search for small molecules that target cellular kinases.

The synthetic molecule that the paper describes—HBS 1—is based on chemically stabilized secondary structure of a protein that is mimicking specific fold, called ?-helix ,- and shows outstanding potential for suppression of tumor growth. This compound was specifically designed to interrupt the type of molecular conversation within cell (called cell signaling) that promotes growth of cancer cells. Creation of HBS 1 required a method for locking correct helical shapes in synthetic strings of amino acids – a method previously developed at NYU.

The studies conducted at NYU and USC show that the molecule disrupted the cancer cell signaling network and reached the correct target in the cell to provide a rapid blockade of tumor growth. Importantly, the compounds did not show any signs of toxicity or negative impact in the test host.

While the in vivo experiments in this research were conducted using renal carcinoma cells, the principles of this design are applicable to many human conditions, including other cancers, cardiovascular diseases, and diabetic complications. The general concept of the study, the interruption of the connection between genes as they conspire to promote cancer growth, is general and applicable to the protein cell to protein cell "conversations" implicated in a host of human diseases.

More information: Protein domain mimetics as in vivo modulators of hypoxia-inducible factor signaling, www.pnas.org/cgi/doi/10.1073/pnas.1312473110

Provided by New York University

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