

## A key link between tumors and healthy tissue identified

March 21 2014, by Bill Hathaway

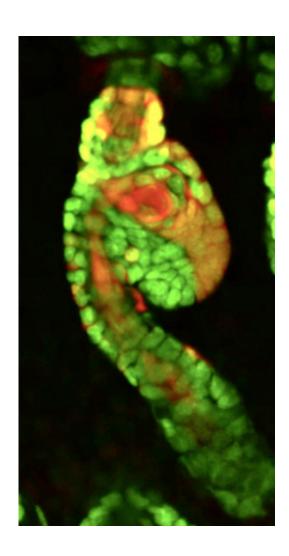


Photo of hair follicle stained to identify beta catenin mutations, which recruit neighboring cells to become hair follicles.



(Medical Xpress)—The delicate balance between development of normal tissue and tumors depends in part upon a key molecular switch within cells, Yale School of Medicine researchers report in the March 21 issue of the journal *Science*. Their findings reveal a potential mechanism used by cancer cells to recruit healthy cells to promote tumor growth and suggest new strategies to generate healthy tissue.

"The fascinating result is that you do not need every cell to be mutated to create tumors, and that individual mutated cells can co-opt their neighbors to fuel tissue growth via this mechanism," said Valentina Greco, assistant professor of genetics and of dermatology at the Yale Stem Cell Center, researcher for the Yale Cancer Center, and senior author of the study.

To understand how <u>healthy cells</u> can be transformed into growth-promoting cells, the Yale team of researchers led by Elizabeth Deschene and Peggy Myung induced new hair growth in mice by activating a mutant form of beta-catenin protein within hair follicle-producing <u>stem cells</u>. These cells recruited normal neighboring cells to become hair follicle cells by secreting a protein called Wnt, which promotes hair growth. Understanding this key interface brings researchers closer to safely leveraging the power of stem cells, which have the unique ability to create a variety of tissue types but are hijacked in many cancers.

"We now know, at the cellular level, how these cells recruit new growth," Greco said. "We are excited to now use this information to investigate how to promote growth in a controlled manner to achieve normal tissue regeneration."

**More information:** β-Catenin Activation Regulates Tissue Growth Non–Cell Autonomously in the Hair Stem Cell Niche, *Science* 21 March 2014: Vol. 343 no. 6177 pp. 1353-1356 DOI: 10.1126/science.1248373



## Provided by Yale University

Citation: A key link between tumors and healthy tissue identified (2014, March 21) retrieved 19 April 2024 from <a href="https://medicalxpress.com/news/2014-03-key-link-tumors-healthy-tissue.html">https://medicalxpress.com/news/2014-03-key-link-tumors-healthy-tissue.html</a>

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