

Overcoming barriers in the quest to starve tumors of blood supply

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One of the most exciting strategies researchers are pursuing for fighting cancer is to cut off the blood supply of cancerous cells. However, many initially-promising therapies have failed in part because tumor cells counteract these therapies by increasing their production of "pro-angiogenic" proteins that promote new blood vessel growth and boost tumor blood supply. In a new study, researchers have found a way to turn the tables on this process by disrupting the ability of vascular endothelial cells (blood vessel-forming cells) to respond to these pro-angiogenic signals from tumors. The findings could open the door to new cancer treatments with a lower risk of drug resistance.

The study was conducted by an international team of researchers from the National Institutes of Health, the Chinese Academy of Sciences and Shanghai Jiao Tong University, and the University of Missouri. Their innovative approach inhibits the replenishment of an intracellular substrate [vascular endothelial cells](#) need to respond to pro-angiogenic signals. As the substrate gets used up, this reduces the ability of endothelial cells to respond to a pro-angiogenic agent called vascular endothelial growth factor, or VEGF, thus limiting the growth of new blood vessels. What's more, when [tumor cells](#) attempt to overcome the therapy by increasing VEGF production, [endothelial cells](#) only consume the substrate faster, enhancing the effectiveness of the therapy and further reducing tumor blood supply. The approach has been successful in initial tests using mice, zebrafish and cell culture.

Brant Weinstein will present this research on Saturday, July 16 from

7:45-8:00 p.m. during the Haematopoiesis and Vascular Biology session
in Grand Ballroom 7B as part of The Allied Genetics Conference,
Orlando World Center Marriott, Orlando, Florida

Provided by Genetics Society of America

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