

Immune system molecule promotes tumor resistance to anti-angiogenic therapy

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A team of scientists, led by Napoleone Ferrara, MD, has shown for the first time that a signaling protein involved in inflammation also promotes tumor resistance to anti-angiogenic therapy.

The findings by Ferrara – professor of pathology at the University of California, San Diego School of Medicine and senior deputy director for basic science at the UC San Diego Moores Cancer Center – and colleagues at Genentech, a biotechnology firm based in South San Francisco, are published in the August 4 Advance Online Publication of the journal *Nature Medicine*.

Angiogenesis is a physiological process in which new blood vessels form from existing vessels. It is fundamental to early development and wound healing, but some [cancer tumors](#) exploit angiogenesis to promote [blood vessel growth](#) and fuel a tumor's transition from a benign to a malignant state.

In the late 1980s, Ferrara led efforts to identify a key gene (VEGF) involved in angiogenesis and subsequent development of the first drugs to block VEGF-mediated growth in a variety of cancers, among them lung, kidney, brain and colorectal. Researchers discovered, however, that similar to other therapies, VEGF-targeting drugs may lose effectiveness as tumors develop resistance, allowing cancers to recur.

The latest research highlights the role of interleukin-17 or IL-17, one of a family of signaling molecules called cytokines that are involved in the body's immune response. Ferrara and colleagues discovered that IL-17 signaling in tumor-infiltrating T cells, part of the body's [adaptive immune response](#), encourages resistance to the VEGF-blockade in mouse models.

"Our work has the potential to have major translational and therapeutic relevance," said Ferrara. "By inhibiting the effects of IL-17 with

[monoclonal antibodies](#) or other blockers, we can potentially improve the clinical efficacy of VEGF-targeting drugs."

More information: *Nature Medicine* [DOI: 10.1038/nm.3291](#)

Provided by University of California - San Diego

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