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Blood vessels contribute to their own growth and oxygen delivery to tissues and tumors (w/ Video)

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Researchers at the University of North Carolina at Chapel Hill School of Medicine and the College of Arts & Sciences have identified a new biological process that spurs the growth of new blood vessels.

Vascular networks form and expand by "sprouting," similar to the way trees grow new branches. The process allows fresh oxygen and nutrients to be delivered to tissues, whether in a developing embryo or a cancerous tumor. Up until now, scientists thought that the molecular signals to form new sprouts came from outside the vessel. But new research from UNC has shown that signals can also come from within the blood vessel, pushing new blood vessel sprouts outward.

The findings, published in the Sept. 15 issue of the journal *Developmental Cell*, could give important insights into the formation of the vasculature needed to feed new tumors.

In experiments using mouse embryonic stem cells and mouse retinas, the researchers found that defects in a protein called Flt-1 lead to abnormal sprouts and poor vessel networks. Other research recently showed that levels of Flt-1 protein are particularly low in the dilated and leaky blood vessels that supply tumors with oxygen.

"The blood vessels themselves seem to participate in the process guiding the formation of the vascular network," said senior study author Victoria

L. Bautch, Ph.D., professor of biology at UNC. "They do not just passively sit there getting acted upon by signals coming from the outside in. Rather, they produce internal cues that interact with external cues to grow."

The growth of new blood vessels can be stimulated by cascades of events within the cell - known as pathways - the most notable of which centers around the three proteins Flt-1, Flk-1 and VEGF. Scientists have known for years that Flk-1 is a positive regulator that responds to VEGF by pulling the emerging sprout outward from its parent blood vessel.

The role of its sister protein Flt-1, however, was not clearly understood. Bautch and colleagues hypothesized that Flt-1 is a negative regulator -- soaking up VEGF molecules so they are not available to interact with Flk-1 and signal for new blood vessels.

The researchers mixed two different types of mouse embryonic stem cells - one batch with normal Flt-1 protein levels, the other with no Flt-1 protein. They found that the genetic makeup of the area at the base of the sprout - rather than at the sprout itself - determined whether the sprout behaved normally or abnormally.

"The cells on each side of sprout produce and send out the soluble form of the protein, blocking the sprout from forming anywhere but in one spot and in one direction," says Bautch. "So when the sprout first forms, instead of flopping back onto its parent vessel, it has a corridor to push it forward away from the parent."

Bautch, who is also a member of the Program in Molecular Biology and Biotechnology, the UNC McAllister Heart Institute and UNC Lineberger Comprehensive Cancer Center, notes that the more scientists understand about the sophistication and complexity of the mechanisms guiding the formation of blood vessel sprouts, the better equipped they will be to

develop therapeutic interventions to produce or to halt new blood vessels .

Source: University of North Carolina School of Medicine (news : web)

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