Scientists identify a key molecule that blocks abnormal blood vessel growth in tumors

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A new and better understanding of blood vessel growth and vascular development (angiogenesis) in cancer has been made possible by research carried out by a team of scientists from Moffitt Cancer Center, the University of Florida, Harvard University, Yale University and the Children's Hospital of Los Angeles.

The research team published the results of their investigation in a recent issue of Proceedings of the National Academy of Sciences.

"Vascular development is a fundamental biological process that is tightly controlled by both pro-and anti-angiogenic mechanisms," said Edward Seto, Ph.D., a co-author of the study and professor and chairman of the Department of Molecular Oncology at Moffitt. "Physiological angiogenesis occurs in adults only under specific settings. Excess angiogenesis contributes to a variety of diseases, including cancer. In cancer, vascular endothelial growth factor (VEGF) is commonly overproduced."

The goal of the research was to determine how angiogenesis is regulated by positive and negative biological activities.

"Understanding the biological principles that direct vascular growth has important clinical implications because cancers are highly vascularized," concluded Seto.

This meant seeking a better understanding of the relationship between the chromatin insulator binding factor CTCF and how it regulates VEGF expression.

"At the heart of vascular development is VEGF which, in precise doses, is an important stimulator of normal blood vessel growth," explained Seto. "However, VEGF - probably the most important stimulator of normal and pathological blood vessel growth - is regulated by a number of factors."

According to Seto, the study suggests that CTCF can block VEGF from being activated. Therefore, targeting CTCF may be an effective way to fine tune VEGF and control angiogenesis. The potential to manipulate CTCF opens a window to regulate VEGF and subsequently, the potential to manage angiogenesis and cancer.

"The real significance of this work has been apparent in experiments done at the University of Florida and at Harvard University, where our colleagues used mouse models to demonstrate that depletion of CTCF produces excess angiogenesis in animals," explained Seto. "Like finding a small key piece in a giant puzzle, it's truly exciting."

Provided by H. Lee Moffitt Cancer Center & Research Institute