Study identifies a key protein in blood vessel growth

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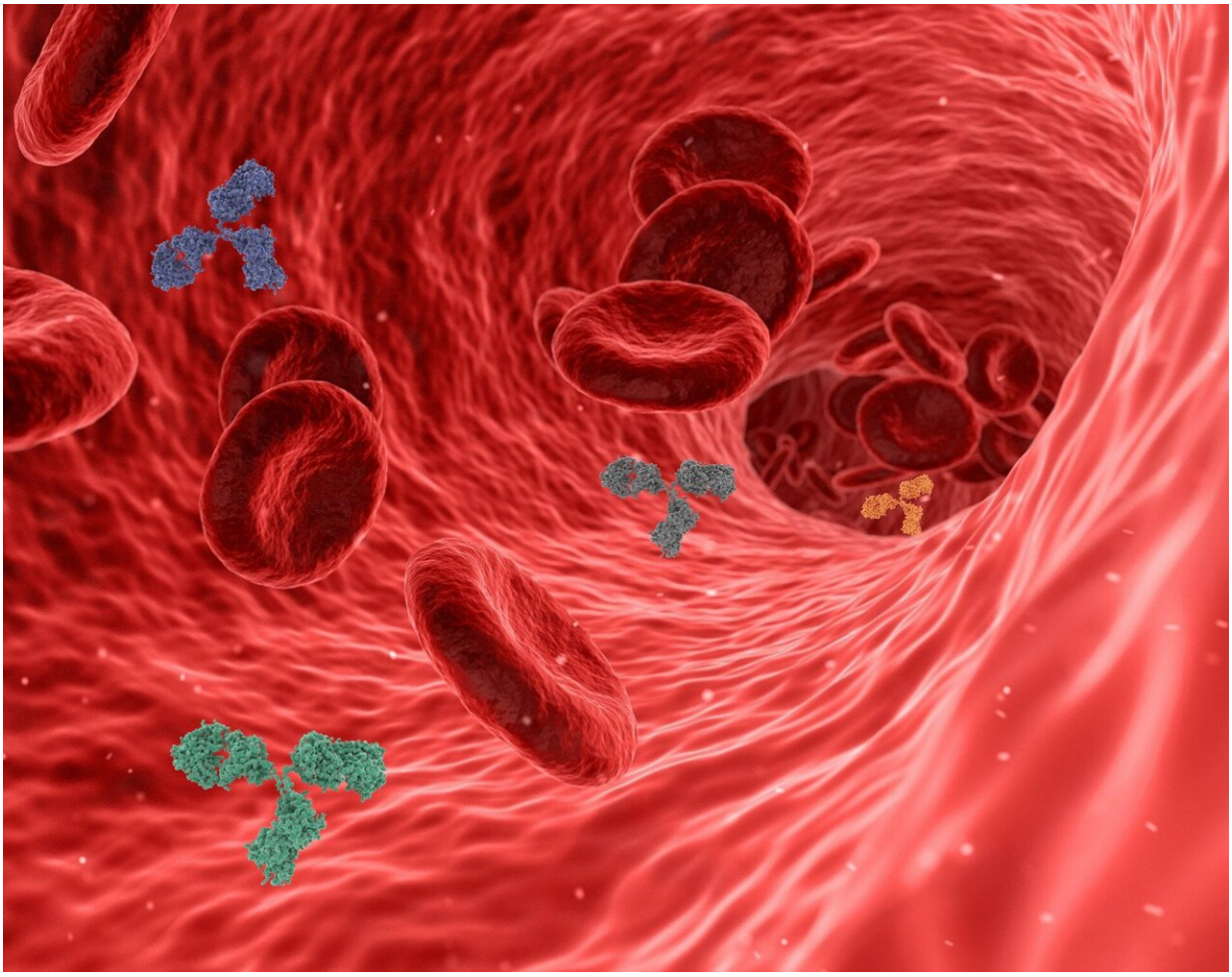

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Blood vessels are responsible for the appropriate and efficient delivery of nutrients and oxygen to the whole body. To do so, they must grow and branch to reach every cell in a process called angiogenesis. The precise regulation of the sprouting and pruning of blood vessels is complex and partly unknown, but endothelial cells, those lining the inner part of the vessels, are known to play an important role.

The growth and proliferation of endothelial cells is promoted by a protein known as mTORC1. Controlling its activity is important for organizing a coherent branching of blood vessels, and alterations in this process may lead to vascular malformations.

New research from the Mariona Graupera's lab (Josep Carreras Leukemia Research Institute), published in *Science Signaling* reveals that PI3K-C2b, a family member of the PI3K kinases, is responsible for mTORC1 fine-tuning through its inhibition.

In a series of experiments using mice models and human cells, researchers found that animals with an inactive form of PI3K-C2a displayed aberrantly enlarged blood vessels. Similarly, when PI3K-C2b was transiently inactivated, endothelial cells appeared larger than usual. Both effects correlated with an increased expression of mTORC1 and were restored upon its external repression.

The findings are important because mutations in components of the PI3K family of proteins are frequent in patients with congenital vascular disorders. Understanding the link between one and the other may be useful in finding new therapeutic targets in the future.

More information: Piotr Kobialka et al, PI3K-C2β limits mTORC1 signaling and angiogenic growth, *Science Signaling* (2023). DOI: 10.1126/scisignal.adg1913

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