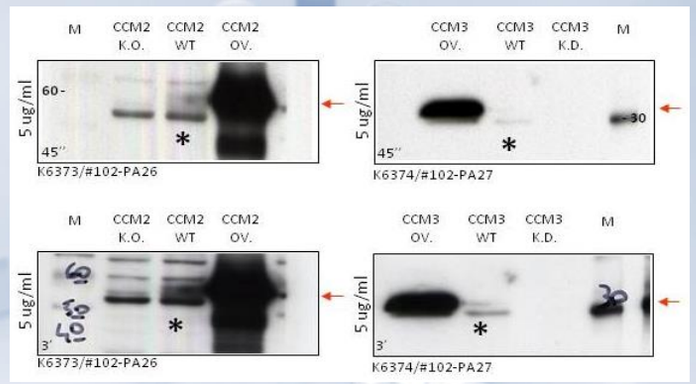

Western Analysis of human CCM2 and human CCM3.
K.O. = knock out (completely deleted)
K.D. = knock down (not completely deleted)
OV. = over expressed in COS1 cells
The experiment was performed by Elisabetta Dejana's group, IFOM-IEO-Campus, Milan Italy

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Release 11/2017

Code generated by ZXing Project

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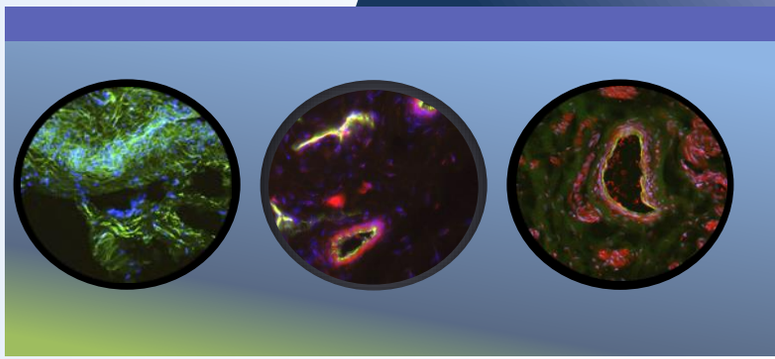
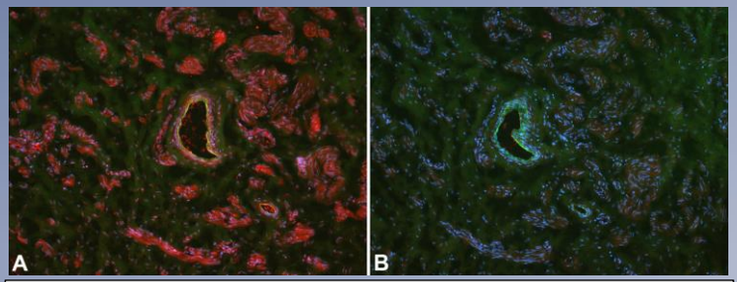


Anti-Human ccbe1 / CCM1-3 antibodies

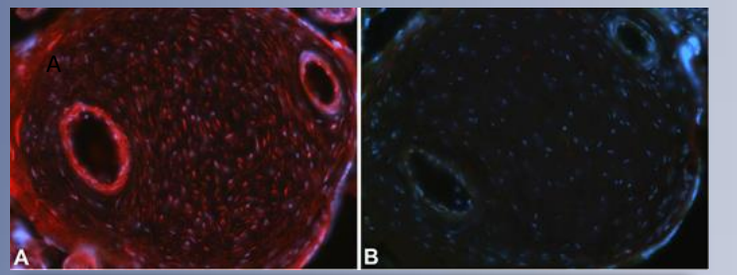
Anti-human ccbe1

The lymphatic system comprises a vascular system separate from the cardiovascular system, essential for immune responses, fluid homeostasis and fat absorption. Lymphatic vessels develop in a complex process termed lymphangiogenesis that involves budding, migration and proliferation of lymphatic endothelial progenitor cells. A few genes, such as FLT4, FOXC2 and SOX18, are known to be critically involved in lymph vessel formation in humans.
Lymphedema, lymphangiectasias, mental retardation and unusual facial characteristics define the autosomal recessive Hennekam syndrome. Homozygosity mapping identified a critical chromosomal region containing ccbe1, encoding Collagen and Calcium-Binding EGF-domain-1, a secreted protein which is required for embryonic lymphangiogenesis in zebrafish.
ccbe1 is not expressed in endothelial cells of lymph vessels, and it may be a component of the extracellular matrix. In zebrafish, ccbe1 expression was observed along the earliest migration routes of endothelial cells that sprout from the posterior cardinal vein and migrate circuitous before developing into lymphatic vessels. ccbe1 might therefore be involved in guidance of these migrating cells.

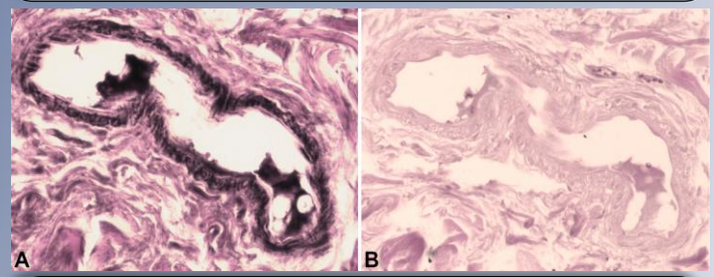


Immunofluorescence staining (red) of ccbe1 in human foreskin tissue using a polyclonal rabbit anti-human ccbe1 antibody [#102-PA36] and counter staining of nuclei with Dapi. The section was fixed with 4% PFA for 25 min, the antibody was diluted 1:100. **B)** Control without primary antibody (yellow in A and green in B correponds to the autofluorescence within the Membrana elastica interna of an artery). A signal is visible in fibrocytes, smooth muscle cells and probably in endothelial cells.
The experiment was performed by the research group of Prof. Dr. J. Wilting, University Göttingen, Germany.

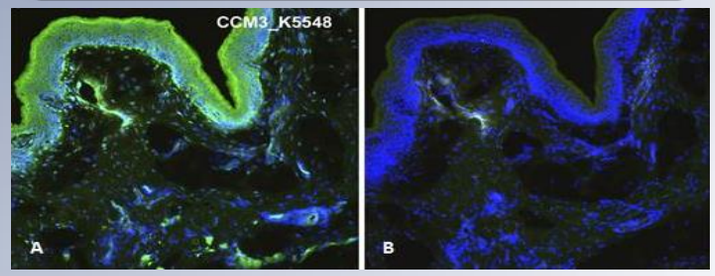
Immunofluorescence staining (green) of human foreskin (cryo-section of unfixed tissue) with anti-human CCM2 (dilution 1:50) **[#102-PA26**]. **A**) Note specific staining in the wall of microvessels. **B**) Negative control of a consecutive section. Note non-specific fluorescence in elastic fibres in the adventitia of an arteriol. Nuclei counter-stained with Dapi (blue).

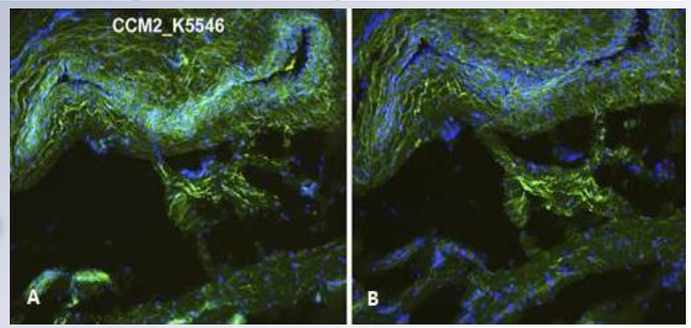


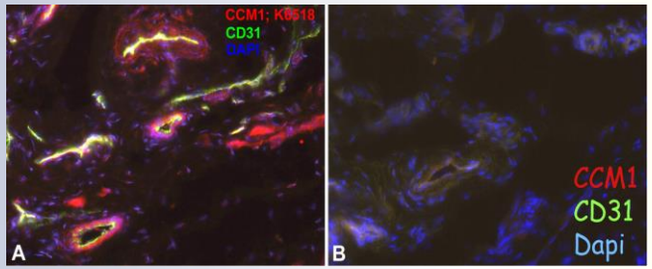
Immunofluorescence staining (red) of ccbe1 in human placenta tissue using a polyclonal rabbit anti-human ccbe1 antibody **[#102-PA36**]. The section was fixed with 4% PFA for 25 min, the antibody was diluted 1:100. **B**) Control without primary antibody. A signal is visible in fibrocytes, smooth muscle cells and probably in endothelial cells.



Immunoperoxidase staining of ccbe1 in human skin using a polyclonal rabbit anti-human ccbe1 antibody [#102-PA36]. The section was fixed with 4% PFA overnight, the antibody was diluted 1:100. **B**) Control without primary antibody. A signal is visible in smooth muscle cells, a little bit weaker in endothelial cells as well as in the connective tissue.
The experiments were performed by the research group of Prof. Dr. J. Wilting, University Göttingen, Germany.







Immunofluorescence staining of human foreskin with anti-CCM1 (dilution 1:50) **[#102-PA25**]. Costaining of endothelial cells with anti-CD31. Note specific staining in the wall of a subset of vessel. Nuclei counter-stained with Dapi. **B**) Control.

Cerebral cavernous malformations (CCMs) are sporadically acquired or inherited vascular lesions of the central nervous system consisting of clusters of dilated thin-walled blood vessels that predispose individuals to seizures and stroke. Mutations in CCM1 (KRIT1), CCM2 (Malcaverin), or CCM3 lead to cerebral cavernous malformations, one of the most common hereditary vascular diseases of the brain. Endothelial cells within these lesions are the main disease compartments. CCM1 stabilizes endothelial junctions and is essential for vascular morphogenesis in mouse embryos. It was shown that CCM1 represents an antiangiogenic protein to keep the human endothelium quiescent. CCM1 inhibits endothelial proliferation, apoptosis, migration, lumen formation, and sprouting angiogenesis in primary human endothelial cells. It also interacts with the CCM2 gene product. Adenoviral CCM3 expression inhibits endothelial cell migration, proliferation, and tube formation while down regulation of endogenous CCM3 results in increased formation of tube-like structures.

Immunofluorescence staining (green) of human foreskin (cryo-section of unfixed tissue) with anti-human CCM3 (dilution 1:50) **[#102-PA27**]. **A**) Note specific staining in the epidermis and in the wall of microvessels. **B**) Negative control of a consecutive section. Nuclei counter-stained with Dapi (blue).
The experiment was performed by the research group of Prof. Dr. J. Wilting, University Göttingen, Germany.

Anti-human CCM1/CCM2/CCM3