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HARNESSING THE PERICYTES TO PROMOTE NEUROVASCULAR REPAIR AFTER ISCHEMIC STROKE

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Ischemic stroke constitute a major cause of death and disability of the adults in the world. Unfortunately, no efficient therapy does yet exist. Endogenous neurovascular restorative responses are triggered within the ischemic tissue, as an attempt from the brain to recover. Ischemic stroke triggers the formation of new microvasculature in the peri-infarct region via activation of various angiogenic mechanisms. Neuronal survival is higher in the tissue undergoing angiogenesis, correlating with longer survival in stroke patients. Angiogenesis is a highly dynamic process that involves close and finely tuned interactions between brain endothelial cells and pericytes. Pericytes play major roles in regulating the cerebral blood flow, angiogenesis, microvasculature stability, and blood-brain barrier (BBB) properties. Ischemic stroke profoundly affects the function of pericytes by triggering their death and detachment from brain endothelial cells, which impairs key neurovascular functions within the ischemic tissue. Using *in vivo* and *in vitro* approaches, our recent work demonstrates that vascular endothelial growth factor isoform-B (VEGF-B), which acts as survival factor, promotes the formation of stable microvasculature within the ischemic tissue by specifically enhancing the survival of pericytes and their interaction with brain endothelial cells. We found that the effects of VEGF-B are mediated via its specific receptor VEGFR-1 that is predominately expressed in brain pericytes. Our study unravelled an unknown role of VEGF-B/VEGFR-1 signalling in rescuing the function of pericytes by inducing expression of the anti-apoptotic protein, B-cell lymphoma 2 (Bcl-2) and AMP-activated protein kinase α (AMPK α) protein, which is involved in energy homeostasis. Moreover, VEGF-B/VEGFR-1 signalling stimulates the release of factors stimulating a reparative angiogenesis that does not compromise microvasculature stability and BBB permeability. Our findings suggest that strategies aiming to stimulate the endothelial cell-pericyte crosstalk constitute a promising therapeutic approach to promote neurovascular repair upon ischemic stroke.

Biography

Ayman ElAli has completed his PhD in 2010 at the University of Duisburg-Essen in Germany. He pursued his Post-doctoral trainings at the research centres of University Hospital of Essen in Germany, and CHU de Québec in Canada. He joined in 2015 the Department of Psychiatry and Neuroscience, Faculty of Medicine, Laval University, Canada, as an Assistant Professor. His research program aims at Investigating Neurovascular Interactions Following Stroke with an Emphasis on Developing New Therapeutic Approaches. He has published more than 37 papers in top-tier journals and has been serving as Referee in several reputed funding organizations and journals.

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