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Synaptic Adhesion Like Molecule (*SALM4*) regulates angiogenic functions via VEGFR2 activation

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Synaptic Adhesion Like Molecules (SALMs) is the adhesion molecules, highly enriched in nervous system, include five members (*SALM 1-5*). All SALMs promote neurite outgrowth, while *SALM4* uniquely increases the number of primary processes extending from the cell body. However, the property of *SALM4* in Endothelial Cell (EC) is still unknown. Here, we discovered that *SALM4* mRNA expression was increased during differentiation from Endothelial Progenitor Cell (EPC) to EC. Unlike other SALMs, *SALM4* was expressed specifically in EC. To find functions of *SALM4*, we performed *in vitro* assays. Wound and chemotactic migration assays showed that knock down of *SALM4* attenuates EC migration. Next, we found tube formation was decreased tube length in *SALM4* deletion EC. EC Survival was reduced in *SALM4* depletion. In mouse organs, *SALM4* shows organ specificity, it was mainly expressed brain and kidney. Consistent with this observation, EC recruitment impaired in *SALM4* KO mice injected matrigel with VEGF. Aortic sprouting reduced in *SALM4* KO mice aorta implanted matrigel. To elucidate the mechanism of *SALM4* under VEGF treatment, we analyzed VEGFR2 activation. Silencing *SALM4* in EC suppressed phosphorylation of VEGFR2. Moreover, downstream of VEGFR2 signaling was also reduced. These results suggest that *SALM4* has a potential role in regulating EC migration via activation of VEGFR2.

Biography

Dong Young Kim is currently pursuing his Doctor's course in Vascular Biology at Yonsei University, Republic of Korea. He has completed his Bachelor's degree in Biochemistry at Yonsei University. His research fields are tumor angiogenesis and identifying angiogenic functions of endothelial cell enriched genes in endothelial cells.

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