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Korean Society of
Lipidology and Atherosclerosis

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▪ Education / Appointments

1992	B.S. Molecular Biology, Seoul National University, Korea
1995	M.S. Life Science, POSTECH, Korea
1998	Ph.D. Life Science, POSTECH, Korea
2008-present	Assistant/Associate Professor, KAIST, Korea

▪ Research Interests

Regulation of Angiogenesis
Cerebrovascular Diseases
Endothelial-pericyte Interaction

▪ Brief List of Publications

1. Lee S, Kim IK, Ahn JS, Woo DC, Kim ST, Song S, Koh GY, Kim HS, Jeon BH and Kim I. Deficiency of endothelium-specific transcription factor Sox17 induces intracranial aneurysm. Circulation 2015;131(11):995-1005.
2. Lee SH, Lee S, Yang H, Song S, Kim K, Saunders TL, Yoon JK, Koh GY and Kim I. Notch pathway targets proangiogenic regulator Sox17 to restrict angiogenesis. Circ Res 2014;115(2):215-226.
3. Yang H, Lee S, Lee S, Kim K, Yang Y, Kim JH, Adams RH, Wells JM, Morrison SJ, Koh GY and Kim I. Sox17 promotes tumor angiogenesis and destabilizes tumor vessels in mice. J Clin Invest 2013;123(1):418-431.
4. Kim I, Saunders TL and Morrison SJ. Sox17 dependence distinguishes the transcriptional regulation of fetal from adult hematopoietic stem cells. Cell 2007;130(3):470-483.

Role of Sox17 in Cerebrovascular System

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transcription factors contribute to endothelial lineage specification during vascular example, Sox17 has been revealed to differentiation of nascent endothelial cells. transcription factors are required for artery adulthood remains unknown. Intracranial characterized by thin wall and ballooning is an arterial vascular disorder, frequently rupture with high mortality. Although risk factors associated with IA have been hereditary basis of IA remains incompletely Therefore, genetically-modified animals accu- IA and related pathogenesis have been drug development has been delayed. factor Sox17 is robustly expressed in cells of normal intracerebral arteries. The combination of *Sox17* deficiency and angiotensin II infusion (for hypertensive stress) in mice induces vascular abnormalities closely resembling the characteristic features of IA, such as luminal enlargement, wall thinning, tortuosity, and subarachnoid hemorrhages. This combination impairs cell-cell junctions, proliferation capacity, and paracrine secretion in endothelial cells of intracerebral arteries, highlighting key endothelial dysfunctions that lead to IA pathogenesis. Moreover, human IA samples showed reduced Sox17 expression and impaired endothelial integrity, further strengthening the applicability of this animal model to clinical settings.

Our findings demonstrate that *Sox17* deficiency in mouse can induce IA under hypertensive stress, suggesting *Sox17* deficiency as a potential hereditary factor for IA formation. The *Sox17*-deficient mouse model provides a novel platform to develop therapeutics for IA.