Murine Model of Intracranial Aneurysm

- Protective Effect of Resveratrol Against Intracranial Aneurysm Rupture in Mice
- Exploration of Immune-Related Transcription Control/Regulation in Intracranial Aneurysm Through KEGG Analysis and in-vivo Validation
- Kisspeptin-10 Prevents the Development of Cerebral Aneurysms by Reducing the Expression of Egr-1
- Enhanced intracranial aneurysm development in a rat model of polycystic kidney disease
- A Modified Mouse Model of Intracranial Aneurysm Based on Hemodynamic Change and Hypertension
- A Preliminary Study of Effect of Melatonin on Inflammation and Hypoxia-Related Factors in a Mouse Model of Elastase-Induced Intracranial Aneurysm
- Identification and Verification of the Driver Genes for the Formation and Development of Intracranial Aneurysms
- Cerebral microvascular density, blood-brain barrier permeability, and support for neuroinflammation indicate early aging in a Marfan syndrome mouse model

Genetic modification in a mouse model of Intracranial Aneurysms, including deletion or overexpression of a particular gene, provides an excellent means for examining basic mechanisms behind disease pathophysiology and developing novel pharmacological approaches. All existing animal models need some pharmacological treatments, surgical interventions, or both to develop IAs, which is different from the spontaneous and natural development of aneurysms under the influence of the classical risk factors. The benefit of such animal models is the development of IAs in a limited time. However, clinical translation of the results is often challenging because of the artificial course of IA development and growth. Khan et al. summarized the continuous improvement in mouse models of IAs. Moreover, they discussed the pros and cons of existing mouse models of IAs and highlighted the main translational roadblocks and how to improve them to increase the success of translational Intracranial Aneurysm research [1].

Intracranial aneurysm rupture causes severe disability and high mortality. Epidemiological studies show a strong association between decreased vitamin D levels and an increase in aneurysm rupture. However, the causality and mechanism remain largely unknown. Kimura et al. tested whether vitamin D deficiency promotes aneurysm rupture and examined the underlying mechanism for the protective role of vitamin D against the development of aneurysm rupture utilizing a mouse model of intracranial aneurysm. Mice consuming a vitamin D-deficient diet had a higher rupture rate than mice with a regular diet. Vitamin D deficiency increased proinflammatory cytokines in the cerebral arteries. Concurrently, vitamin D receptor knockout mice had a higher rupture rate than the corresponding wild-type littermates. The vitamin D receptors on endothelial and vascular smooth muscle cells, but not on hematopoietic cells, mediated the effect of aneurysm rupture. The results establish that vitamin D protects against the development of aneurysmal rupture through the vitamin D receptors on vascular endothelial and smooth muscle cells. Vitamin D supplementation may be a viable pharmacologic therapy for preventing aneurysm rupture [2]

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Khan D, Li X, Hashimoto T, Tanikawa R, Niemela M, Lawton M, Muhammad S. Current Mouse Models of Intracranial Aneurysms: Analysis of Pharmacological Agents Used to Induce Aneurysms and Their Impact on Translational Research. J Am Heart Assoc. 2024 Jan 23:e031811. doi: 10.1161/JAHA.123.031811. Epub ahead of print. PMID: 38258667.

2)

Kimura T, Rahmani R, Miyamoto T, Kamio Y, Kudo D, Sato H, Ikedo T, Baranoski JF, Uchikawa H, Ai J, Lawton MT, Hashimoto T. Vitamin D deficiency promotes intracranial aneurysm rupture. J Cereb Blood Flow Metab. 2024 Jan 19:271678×241226750. doi: 10.1177/0271678×241226750. Epub ahead of print. PMID: 38241458.