

## Abstract View

### **DEFICIENCY OF COX-1 GENE EXPRESSION IN MICE REDUCES THE INFARCTION VOLUME IN A 24-HOUR PERMANENT FOCAL CEREBRAL ISCHEMIA MODEL**

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Inflammatory reactions have an important role in the ischemic pathophysiology.

Cyclooxygenase (COX) is the key enzyme in converting arachidonic acid to prostanoids that, in turn, are the major contributors to the intrinsic inflammatory response. In this study, we used COX-1-null mice to investigate the role of COX-1 gene expression in a permanent focal cerebral ischemia model. Adult littermates (wild type +/+, heterozygous +/-, and homozygous -/-), were used. Genetic status was determined using a PCR analysis. The mice were anesthetized with chloral hydrate (350 mg/kg, IP). Right femoral artery was cannulated for monitoring of arterial blood pressure and heart rate. Rectal temperature was kept normal throughout anesthesia. Cerebral blood flow was monitored by Laser-Doppler Flowmetry. Permanent focal cerebral ischemia was achieved by passing a monofilament suture via the right external and internal carotid arteries to occlude the right middle cerebral artery (MCA) at its origin. Mice were killed by decapitation at 24 hours after MCA occlusion. The brains were cut into 2-mm coronal slices for staining with 2% tetrazolium chloride to reveal the infarct. The infarction volume was quantified using computer-assisted image analysis. Relative infarction volumes were, in mean  $\pm$ SEM, 42.67  $\pm$ 3.72% (n=6) in COX-1 +/+ mice, 25.97  $\pm$ 8.48% (n=6) in COX-1 +/- mice, and 28.13  $\pm$ 6.22% (n=6) in COX-1 -/- mice, respectively. There was no significant difference in physiological parameters and cerebral perfusion among the groups. Our results demonstrated that lack of COX-1 gene expression might reduce infarction volume in a 24-hour permanent MCA occlusion model in mice.