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RUNX1 expression is upregulated after middle cerebral artery occlusion in rats

Abstract:

Ischemic stroke is a leading cause of death worldwide. With high mortality and morbidity, ischemic stroke results in immense public healthcare burden and serious socioeconomic consequences. Effective pharmacological treatments are urgently required to mitigate the effects of stroke. Runt-related transcription factor-1 (RUNX1), a member of the core-binding factor family of transcription factors, represents a potential therapeutic target for ischemic diseases. RUNX1 is classically considered as the master regulator of developmental hematopoiesis because of its indispensable role in the specification of the hematopoietic lineage during embryogenesis. Whilst the focus of RUNX1 research has predominately been in the hematopoietic field, recent evidence reveals emerging functions of RUNX1 in different tissues under pathological conditions. In the heart, growing evidence showed that RUNX1 expression is increased following myocardial infarction and it negatively correlates with cardiac function. Our previous study demonstrated that antagonizing RUNX1 function reduces infarct size and preserves myocardial contractility following myocardial infarction. In the brain, the role of RUNX1 after cerebral infarction merits an investigation. The present study sought to investigate the expression pattern and function of RUNX1 using a rat model of middle cerebral artery occlusion (MCAO). Rats were subjected to MCAO by means of surgically inserting a monofilament into the middle cerebral artery. We report that RUNX1 expression is increased in the brain after MCAO, and the RUNX1 expression is associated with increased infarct size. On-going study examines whether the increase of RUNX1 can be therapeutically targeted to reduce infarct size after MCAO. Our results suggest the translational potential of RUNX1 as a novel therapeutic target for brain protection after ischemic stroke

Biography

Weihong He is a principal investigator and associate professor at the Department of Physiology, West China School of Basic Medical Sciences and Forensic Medicine, Sichuan University. Weihong obtained an MD (2012) at West China School of Medicine & West China Hospital, Sichuan University, and completed a PhD (2017) at the BHF Glasgow Cardiovascular Research Centre, University of Glasgow. Weihong was associate professor at Jining Medical University (2018-2020). Since 2020, Weihong has led a research group to study the pathophysiology of cardiovascular diseases and to investigate novel therapeutic drugs for the treatment of myocardial infarction and cerebral infarction at Sichuan University. He also teaches physiology and mentors both national and international students. Weihong has expertise in a number of methodologies which span the level of biochemistry, cell biology, isolated organ, and whole animal in vivo disease models