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[관심분야]

Myocardial Infarction, Stroke and Inflammation, Atherosclerosis, ROS and mitochondria

[논문]

1. TK Kim, S Jeon, et al. 2'-5' oligoadenylate synthetase-like 1 (OASL1) protects against atherosclerosis by maintaining endothelial nitric oxide synthase mRNA stability. *Nat Commun.* 2022 Nov 4;13(1):6647
2. S Jeon, et al. Anti-inflammatory actions of soluble Ninjurin-1 ameliorate atherosclerosis. *Circulation* 2020 Nov 3;142(18):1736-1751
3. TJ Yun, et al. Indoleamine 2,3-Dioxygenase-Expressing Aortic Plasmacytoid Dendritic Cells Protect against Atherosclerosis by Induction of Regulatory T Cells. *Cell Metabolism.* 2016 May10;23(5):852-866
4. JH Choi, C Cheong, et al. Flt3 signaling-dependent dendritic cells protect against atherosclerosis. *Immunity* 2011 Nov23;35(5):819-831
5. HJ Jeon, JH Choi, I Jung, et al. CD137(4-1BB) Deficiency Reduces Atherosclerosis in Hyperlipidemic Mice. *Circulation* 2010 Mar 9;121(9):1124-1133

Heart-Immune-Brain network in the pathogenesis of cardio-cerebrovascular disease

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This lecture amalgamates insights from the studies to unveil the intricacies of bidirectional interactions within this physiological axis and their profound implications for cardio-cerebrovascular diseases. The research investigates the dynamic interplay between the cardio-immune and neuro-immune systems, unravelling their roles in heart-to-brain and brain-to-heart interactions. Employing a mouse model of myocardial infarction, the study elucidates how cardiac dysfunction instigates changes in the cardio-immune system, subsequently leading to alterations in brain function. Conversely, in a stroke model, the study explores the neuro-immune system's influence on cardiac function, shedding light on connections between stroke-induced macrophage production and cardiac performance. Additionally, the analysis of extracellular vesicles (EVs) secreted in response to cardiac dysfunction provides novel insights into the communication between the heart and brain.

The studies focus on myeloid cell heterogeneity, delineating the transition from local tissue-resident macrophage proliferation to circulating cell recruit-

ment and phenotypic plasticity. Through single-cell RNA sequencing, a distinct microglia type, denoted as stroke-associated microglia (SAM), emerges. SAM exhibits enhanced antioxidant function and unique molecular markers, proving crucial in mitigating reactive oxygen species (ROS) damage during ischemia/reperfusion (I/R) in the brain. The indispensable role of Peroxiredoxin-1 (Prdx1) in SAM's antioxidative capacity is highlighted, influencing ROS defense genes and promoting stroke-protective molecules.

This comprehensive exploration of the heart-immune-brain network provides a paradigm shift in understanding cardio-cerebrovascular diseases. The elucidation of molecular and functional immune cell mechanisms not only facilitates the development of innovative therapeutic strategies but also introduces potential targets for intervention. By unraveling the complexities of this network, this lecture contributes to groundbreaking research and the cultivation of future researchers poised to lead advancements in science and technology.