

## JOINT EVENT

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**Fimasartan protect cardiac myocyte damage in ischemic heart diseases****Dong Ju Choi<sup>1</sup>, Byung-Kwan Lim<sup>2</sup> and Jin Joo Park<sup>1</sup>**<sup>1</sup>Seoul National University Bundang Hospital, South Korea<sup>2</sup>Jungwon University, South Korea

Fimasartan is a new angiotensin II receptor antagonist (ARB) with high selectivity for the AT1R subtype. Fimasartan showed superior inhibitory activity in the contraction of isolated rabbit thoracic aorta compared with other ARBs and showed protective effect in acute MI model. Fimasartan significantly reduced the infarct size after ischemia and reperfusion (I/R) injury and prevented mitochondrial dysfunction through inhibition of Ca<sup>2+</sup> overload during acute reoxygenation. However, it is unknown whether fimasartan has any protective effect on cardiac remodeling after MI. Angiotensin receptor blocker (ARB) treatment before the onset or during the acute stage of myocardial infarction (MI) mitigates the progression of post-MI cardiac remodeling. We investigated the effect of fimasartan, a new ARB, on cardiac remodeling after MI. Sprague-Dawley rats were assigned in to three groups; surgery only (Sham group, n=8), MI without (MI group, n=11), and MI with fimasartan treatment group (MI+fimasartan group, n=14). MI was induced by permanent ligation of the left anterior descending artery (LAD). Treatment with fimasartan (10 mg/kg) was initiated 24 hours after MI and continued for seven weeks via oral administration. After harvesting the heart, the size of infarct and fibrosis area was lower in MI+fimasartan than MI group. MI+fimasartan group had higher left ventricular ejection fraction (LVEF) (66.3±12.5% vs. 51.3±14.8%, P=0.002) and Left ventricular end-diastolic diameter (LVEDD) (9.14±1.11 mm vs. 9.91±1.43 mm, P=0.045) than MI group at seven weeks. In microarray analysis, fimasartan treatment dramatically decreased genes related with inflammatory signal and increased those related with lipid metabolism. In addition, mitochondria membrane ion transport and damage response genes were increased. Fimasartan attenuates the left ventricular (LV) remodeling and cardiac dysfunction after MI. Fimasartan may be used to mitigate the progression of heart failure after MI.