## S50-5 New pharmacological action of beta-blockers

◯Michio NAKAYA[1], Satsuki CHIKURA[1], Motohiro NISHIDA[1], Hitoshi KUROSE[1]
[1]Grad. Sch. of Pharm. Sci., Kyushu Univ.

Heart failure is a clinical state that is not specific disease. The heart of heart failure cannot supply enough amount of blood (oxygen) that peripheral tissues need. Heart failure patients are treated by several drugs such as angiotensin converting enzyme inhibitor and diuretics. It has been recently demonstrated that β-blockers are very effective for treatment of heart failure. They are believed to be effective by blocking excessive stimulation of catecholamine, and reduce oxygen assumption of the heart. It has been reported that a β2-selective blocker ICI-118551 activates cellular signaling through G protein-independent but β-arrestin-mediated pathway. The roles of β-arrestins are established as a component of machinery in cellular desensitization by binding to phosphorylated receptors and inhibiting G protein activation. However, β-arrestins are recently recognized as a scaffold protein as β-arrestins can bind several signaling proteins. Various β-blockers are classified based on several properties: selectivity, duration of action, partial agonist activity, hydrophobicity, and so on. In addition to these properties, β-blocker-induced activation of intracellular signaling though β-arrestin will be another property to differentiate various β-blockers. We would like to show β-arrestin-mediated pharmacological action of β-blockers that are used in cardiovascular diseases.