accelerate the age-related RILA stiffness occurs earlier than to accelerate the age-related RAA stiffness. The mechanism is probably associated with the up-regulated level of RAGE in IRSA media, while the AGES in serum or IRSA media may be involved in the late stage.

**GW26-e4656**

**Polymorphism of RBP4 Locus Is Associated with 5-Year Survival in acute coronary syndrome after coronary revascularization**

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**OBJECTIVES** The rs7094671 was single nucleotide polymorphism of RBP4 locus that was associated with prevalence of coronary artery disease. No data concerning their association with long term prognosis after myocardial infarction is available. The aim of our study was to investigate the association of the RBP4 locus with 5-year overall mortality in patients with acute coronary syndrome after coronary revascularization.

**METHODS** Cohort study included 292 patients with acute coronary syndrome treated with primary PCI, followed for up to 5 years. Genotyping was performed with high resolution melting (HRM) analysis. The analysis was total sample of 292 patients. Simultaneously, changes in the expression levels of phosphorylated extracellular signal-regulated kinase (p-ERK2) in the cardiomyocytes were assessed.

**RESULTS** The cell size of the AngII - treated cardiomyocytes was significantly larger than that of the untreated cardiomyocytes. The expression of hypertrophic markers and p-ERK2, the cell surface area and the [3H] Leucine incorporation rate in the AngII-treated rat cardiomyocytes were detected following RNA interference. Simultaneously, changes in the expression levels of phosphorylated extracellular signal-regulated kinase (p-ERK2) in the cardiomyocytes were assessed. Results:

**CONCLUSIONS** AngII induces hypertrophy in cardiomyocytes and mAKAPβ is possibly involved in this process. The effects of mAKAPβ on AngII-induced cardiomyocyte hypertrophy may be associated with p-ERK2 expression.

**GW26-e4672**

**The role of mAKAPβ in the process of cardiomyocyte hypertrophy induced by angiotensin II**

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**OBJECTIVES** Angiotensin II (AngII) is the central product of the reninangiotensin system (RAS) and this octapeptide contributes to the pathophysiology of cardiac hypertrophy and remodeling. mAKAPβ is an Akinase anchoring protein (AKAP) that has the function of linking to the regulatory subunit of protein kinase A (PKA) and to confining the holoenzyme to discrete locations within the cell.

In this study, we aimed to investigate the role of mAKAPβ in AngII-induced cardiomyocyte hypertrophy and the possible mechanisms involved.

**METHODS** Cultured cardiomyocytes from neonatal rats were treated with AngII. Subsequently, the morphology of the cardiomyocytes was observed and the expression of mAKAPβ and cardiomyocyte hypertrophic markers was measured. mAKAPβ-shRNA was constructed for RNA interference; the expression of mAKAPβ and hypertrophic markers, the cell surface area and the [3H] Leucine incorporation rate in the AngII-treated rat cardiomyocytes were detected following RNA interference. Simultaneously, changes in the expression levels of phosphorylated extracellular signal-regulated kinase (p-ERK2) in the cardiomyocytes were assessed.

**RESULTS** The cell size of the AngII - treated cardiomyocytes was significantly larger than that of the untreated cardiomyocytes. The expression of hypertrophic markers and p-ERK2, the cell surface area and the [3H] Leucine incorporation rate in the AngII-treated rat cardiomyocytes were detected following RNA interference. Simultaneously, changes in the expression levels of phosphorylated extracellular signal-regulated kinase (p-ERK2) in the cardiomyocytes were assessed. Results:

**CONCLUSIONS** AngII induces hypertrophy in cardiomyocytes and mAKAPβ is possibly involved in this process. The effects of mAKAPβ on AngII-induced cardiomyocyte hypertrophy may be associated with p-ERK2 expression.