GW26-e1803
Enhanced Expression of A-Disintegrin-and-Metalloproteinase-17 Promotes Extracellular Matrix Remodeling in Rats With Myocardial Infarction
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OBJECTIVES This study aimed to investigate the effects of dynamic expression of cardiac tissue A-Disintegrin-and-Metalloproteinase-17 (ADAM-17) on myocardial remodeling after myocardial infarction (MI).

METHODS Forty adult male Wistar rats undergoing MI operation were divided into four subgroups based on survival time: MI0d (at the end of the first day), MI1w (at the end of the first week), MI1d (at the end of the fourth week) and MI12w (at the end of the 12th week). Hemo- dynermomeics in each group were performed. Protein expressions of tumor necrosis factor-α (TNF-α), ADAM17 and tissue inhibitor of metalloproteinases-3 (TIMP-3) in ischemic cardiac tissue were analyzed by Western blot.

RESULTS One week after MI, left ventricular weight index (LVWI) was increased and systolic function examined by echocardiography was sharply worsened compared with that in ConiW subgroup (P < 0.05). A red blood cell index and increased collagen accumulation were also displayed in the healing myocardium especially followed by a deter- riorated MI-induced cardiac remodeling. TNF-α level was lower in the MI12w group compared with that in MI0d subgroup (P < 0.05). However, ΔTNF-α level, the net change of TNF-α concentration between the MI and the ConiW groups was significantly increased (P < 0.05). ADAM17 mRNA expression was significantly increased, especially at the end of the 1st week after MI (P < 0.05). The results of Western blot also reveals that the protein expressions of ADAM17 and TIMP-3 were significantly up-regulated, and the expression of TIMP-3 was simultaneously sharply decreased in the MI12w subgroup (P < 0.05).

CONCLUSIONS Enhanced ADAM17 expression may participate in myocardial remodeling, especially in the early stage after MI.

GW26-e2206
Phosphatidylinositol 4-Kinase β (PI4Kβ) and Phosphatidylinositol 4,5-Bisphosphate (PIP2) Modulated the Expression and Trafficking of BKCa Channels
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OBJECTIVES Phosphatidylinositols (PI) play crucial roles in regulating cytokskeletal organization and trafficking of membrane protein. PI metabolism is controlled by phosphatidylinositol kinases. Phosphyd- atidylinositol 4-kinase β (PI4Kβ)-mediated phosphatidylinositol 4,5-biphas- phase (PIP2) production has been suggested to regulate biosynthetic traffic in yeast and mammalian cells. Here, we investigated the effect of PI4Kβ and PIP2 on the expression and trafficking of large conductance calcium activated potassium (BKCa) channels, the key modulator of the tone of vascular smooth muscles, expressing in HEK293 cells.

METHODS HEK293 cells with expressing BKCa channels α subunit (hSlo1) were constructed with transfection. BKCa currents were recording by patch clamp technique under whole-cell and inside-out configuration. Western Blotting, flow cytometry (FCM), confocal were used to investigate the expression and trafficking of BKCa channels. The direct interaction between phosphoinositides and BKCa channels was investigated with PIP strips.

RESULTS PI4Kβ increased the total and membrane expression of BKCa channels expressing in HEK293 cells. The wild type PI4Kβ (PI4KβWT) increased the expression of BKCa channels, while the dominant negative kinase-dead PI4Kβ (PI4KβKD) decreased the expression of BKCa channels compared with the control group. Wortmannin and PAO, the inhibitors of PI4Kβ, decreased the current density under whole-cell configuration. PIP2 increased total open probability (NPO) under inside-out configuration. In PIP2 on the expression and trafficking of BKCa channels. The direct interaction between two phosphoinositides and BKCa channels was investigated with PIP strips.

CONCLUSIONS These data above suggested that PI4Kβ increased the expression and trafficking of BKCa channels to membrane. PI4Kβ may control the PI metabolism and the production of downstream Pls such as PIP2 in order to modulate the expression and trafficking of BKCa channels indirectly.

GW26-e2335
The Construction of Nano-Protein Complexes and Induce Human Bone Marrow-Mesenchymal Stem Cells (hBMSCs) Differentiate Into Cardiac Progenitor Cells (CPCs) by Direct Reprogramming
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OBJECTIVES To explore the best protein modified condition and influence factors to construct nano-protein complexes and preliminarily analyze their process of entering into cells and intracellular meta- bolism. Then we try to induce hBMSCs toregroup into CPCs by using this protein transfection technology, which shows high efficiency and low cytotoxicity to cells. It opens up new way for clinical cardiac reparations and regeneration treatment after myocardial infarction.

METHODS 4 kinds of cardiac specific transcription factors (Tbx5, Hand2, Mef2c, Gata4) are expressed and purified in an improved producing way, we’ve try to synthesis several compact and tightening...