GW26-e1438
Ginsenoside Rb1 Reverses Human Umbilical Vein Endothelial Cells Senescence Induced by Hydrogen Peroxide
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OBJECTIVES To investigate the effect of Ginsenoside Rb1 on human umbilical vein endothelial cells (HUVECs) senescence established by hydrogen peroxide (H2O2).

METHODS Senescence model was established by treating HUVECs with 60 μmol/ L H2O2 for 48 h. The parameters were detected to demonstrate the effect of Ginsenoside Rb1 on senescence and the mechanism involved was also investigated.

RESULTS Compared with the control group, after treated with 60 μmol/ L H2O2, the number of SA β-gal positive cells and the expression of PAI-1 significantly increased. Meanwhile, MDA concentration increased but the expression of SOD1 decreased in the senescent cells. However, after pretreatment of 20 μmol / L Ginsenoside Rb1, the ratio of SA β-gal positive cells, the expression of PAI-1 and the MDA concentration decreased significantly, but the expression of SOD1 increased.

CONCLUSIONS Ginsenoside Rb1 could reverse HUVECs senescence induced by H2O2. The anti-peroxide effects of Ginsenoside Rb1 appears to be particular importance.

GW26-e2240
The inhibitory effect of catestatin on rat's cardiac function was partly mediated by nitric oxide
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OBJECTIVES To investigate the effect and signaling mechanism of catestatin (CST) on cardiac function in normal and heart failure rats.

METHODS Catestatin (CST) on cardiac function in normal and heart failure rats. To investigate the effect and signaling mechanism of catestatin (CST) on cardiac function in normal and heart failure rats. The inhibitory effect of catestatin on rat's cardiac function was dose dependent, L-NAME could inhibit this effect, so the mechanism of CST on normal and heart failure rat's cardiac function was partly mediated by nitric oxide.

GW26-e1019
Role of group I metabotropic glutamate receptors, mGluR1/mGluR5, in connexin43 phosphorylation and inhibition of gap junctional intercellular communication in H9C2 cardiomyoblast
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OBJECTIVES 1. To explore the role of group I metabotropic glutamate receptors, mGluR1/mGluR5, in connexin43 (Cx43) phosphorylation and inhibition of gap junctional intercellular communication (GJIC); 2. To further study the molecular mechanism of role of mGluR1/5 in Cx43 phosphorylation and GJIC inhibition.

METHODS After documenting the presence of mGluR1 and mGluR5 in H9c2 cardiomyoblast cells, addition of the selective mGluR1/5 agonist (S)-3,5 dihydroxyphenylglycine hydrate (DHPG) induced Cx43 phosphorylation and GJIC inhibition in both concentration- and time-dependent manner. The effects of DHPG were abolished by the mGluR1 antagonist LY367385 and the specific inhibitor of MEK1, PD98059 which also reduced phosphorylation of extracellular-signal-regulated protein kinase 1/2 (ERK1/2); but not by the mGluR5 antagonist 6-methyl-2-(phenylethynyl) pyridine hydrochloride (MPEP) or the selective inhibitor (GF109203X) of protein kinase C (PKC).

RESULTS 1. The specific 142 and 130 kDa bands corresponding to molecular weights of mGluR1 and mGluR5, respectively, were detected; 2. The administration of DHPG can induce a relative increase in Cx43-P2 band level and reduced Cx43-P0 and Cx43-P1 band levels. In addition, total amount of Cx43 protein was reduced gradually compared to that of group NC with increasing DHPG concentration. GJIC was inhibited obviously by DHPG compared with that of negative control when Cx43 phosphorylation was most obvious; 3. Preincubation of cells with LY367385 before application of DHPG produced a marked inhibition of agonist-induced Cx43 phosphorylation activation and inhibition of GJIC, however, MPEP pretreatment failed to block them. These data indicate that Cx43 phosphorylation and GJIC inhibition is mediated mainly by mGluR1; 4. Pre-incubation of GF109203X at increasing concentrations had little or no effect on DHPG-induced phosphorylation level of ERK1/2 and Cx43. Meanwhile, the GF109203X even at increasing concentrations was unable to affect DHPG-induced GJIC drop; 5. DHPG-induced Cx43 phosphorylation was attenuated significantly by exposure of cells to increasing concentrations of PD98059, while DHPG-induced phosphorylation level of ERK1/2 also decreased by application of PD98059. PD98059 also blocked the DHPG-induced drop of GJIC with increasing concentrations.

CONCLUSIONS 1. It has been proved that the presence of mGluR1 and mGluR5 in H9c2 cardiomyoblast cells; 2. This study indicates that group I mGluRs induce Cx43 phosphorylation, as shown in Western blot analysis, which contributes to decreased GJIC; 3. The receptor that plays a major role in DHPG-induced Cx43 phosphorylation and GJIC inhibition is mGluR1; 4. PKC pathway is not involved in the process of DHPG-induced Cx43 phosphorylation and GJIC inhibition; 5. These findings confirm in the rat cardiomyoblast cell line H9c2 that molecular mechanisms of DHPG-induced Cx43 phosphorylation are consistent with DHPG- induced inhibition of GJIC via ERK1/2.