Conclusion Pretreatment with the AT1 receptor blocker valsartan can attenuate left ventricular remodelling and failure in a rat model of adriamycin-induced dilated cardiomyopathy by upregulating the expression of angiotensin-converting enzyme 2 and angiotensin-(1–7).

e0139 THE CARDIOMYOGENIC POTENTIAL OF CARDIAC STEM CELLS IN AN IN VITRO COCULTURE SYSTEM

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Features so far documented in reliably isolated and manipulated cardiac stem cell (CSCs) resident within the cardiac tissue suggest that innovative treatments to repair damaged myocardium could have promising clinical applications. However, the capability of resident cardiac stem cell (CSCs) to differentiated into newly formed cardiomyocyte is still controversial. In this study, the cardiomyogenic potential of c-kit+ CSCs isolated from normal adult mouse hearts was evaluated in an in vitro co-culture system.
Methods Magnetic activated cell sorting (MACS) was used to prepare c-kit+ cells from the hearts of ACT-EGFP/MHC-nLAC double transgenic mice. These animals exhibit widespread enhanced green fluorescent protein (EGFP) expression and cardiomyocyte-restricted nuclear b-galactosidase activity, thus permitting simultaneous tracking of cell survival and differentiation. The c-kit+ cells were cocultured with feeder layer neonatal rat cardiomyocytes (NRCMs) for 7 days. Confocal and fluorescence microscopes were used to quantify the differentiation rate of c-kit+ cells in the immunostained cocultures.
Results A subset of the c-kit+ cells underwent cardiomyogenic differentiation when cocultured with NRCMs (0.15% out of all 70, 747 EGFP+ cells screened), but not when cultured alone or when cocultured with mouse fibroblasts (0.00% of the EGFP+ cells screened). The newly formed cardiomyocytes were EGFP+, nuclear b-galactosidase+ and a-actinin+ with clear sarcomere structure, indicating mature functional properties.
Conclusion Normal adult c-kit+ CSCs are able to differentiate into functional cardiomyocytes, but it is a rear event at least in the in vitro coculture system.

e0140 ENDOPLASMIC RETICULUM STRESS INDUCED-APOPTOTIC MODEL BY TUNICAMYCIN IN CULTURED NEONATAL RAT CARDIOMYOCYTES

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Objective To establish endoplasmic reticulum stress induced-apoptotic model by tunicamycin in cultured neonatal rat cardiomyocytes.
Methods Neonatal rat cardiomyocytes in primary culture were exposed to tunicamycin of different concentrations. MTT assay and flow cytometry analysis were applied to measure cardiomyocyte viability. Western blot was used to examine the expression levels of GRP78 and CHOP.
Results Cell viability was time and concentration-dependently decreased. The treatment of tunicamycin produced 42.8±5.8% of apoptotic population in cardiomyocytes. The levels of GRP78 and CHOP significantly upregulated at 6 h. After tunicamycin treatment for 24 h, the upregulation of GRP78 and CHOP reached the maxium.
Conclusion We successfully constructed tunicamycin-induced apoptotic model in cultured neonatal rat cardiomyocytes. The optimal concentration and time of tunicamycin treatment was 100 ng/ml, 72 h, respectively.

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e0141 THE EFFECTS AND MECHANISMS BETWEEN DIDS AND EDRV ON ACUTE ISCHAEMIA-REPERFUSION INJURY MYOCARDIUM

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Aim To investigate the effects and Mechanisms of chloride channel inhibitor, 4, 4′- Diisothiocyanostilbene-2, 2′- disulfonic acid (DIDS) and free radical scavenger, Edaravone (EDRV) on myocardial ischaemia/reperfusion injury (I/RI)in vivo.
Methods Male Sprague-Dawley rats, subjected to 30 min of myocardial ischaemia and 4 h of reperfusion, were divided into five groups: sham group, I/RI group, DIDS group, EDRV group and DIDS+EDRV group. Left ventricular systolic pressure (LVSP), The maximal first derivative of developed pressure (±dp/dtmax), Myocardial infarction size, serum creatine kinase (CK) activity, lactate dehydrogenase (LDH) activity, superoxide dismutase (SOD) activity, malondialdehyde (MDA) concentration, myocardial apoptotic index, reactive oxygen species (ROS) were detected.
Results There were no statistical difference in heart rate in each animal suffering myocardial ischaemia/reperfusion compared with sham group ($p>0.05$, n=8). LVSP and $\pm dp/dt_{max}$ were decreased during the period of myocardial ischaemia except sham but there was no statistical difference ($p>0.05$, n=8), However, following reperfusion, the data showed that DIDS and EDRV significantly improved myocardial function in I/RI rats (n=8, $p<0.05$) and DIDS +EDRV combined administration had a much stronger cardioprotective effect than DIDS or EDRV did alone (n=8, $p<0.05$); the levels of activity of serum creatine kinase (CK) (n=8), lactate dehydrogenase (LDH) (n=8), myocardial infarction size (n=8), myocardial apoptotic index (n=6)showed that there were no statistical difference was observed between DIDS and EDRV groups, DIDS+EDRV treatment further decreased compared with DIDS or EDRV treatment alone ($p<0.05$) for the above-mentioned results; and the levels of the concentration of malondialdehyd (MDA), superoxide dismutase (SOD), reactive oxygen species (ROS), O^{2-} and OH·showed that DIDS reduces free radical weaker than EDRV ($p<0.05$, n=8), and DIDS+EDRV combined administration had a stronger cardioprotective effect than DIDS or EDRV did alone and combined administration possesses synergies action ($p<0.05$, n=8).
Conclusion 1. DIDS and EDRV protect myocardium from MI/R injury via improving cardiac function, reducing infarct size and suppressing cardiomyocyte apoptosis; 2. The mechanisms of cardioprotective effects of DIDS and EDRV were involved in inhibition of ROS activity. The protective effect of combined administration can be further enhanced, suggesting DIDS protects ischaemia/reperfusion injury myocardium via other distinctive mechanisms except above.

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