

[gw22-e0246]

DIDS EXERTS A CARDIOPROTECTIVE EFFECT ON TUNICAMYCIN-INDUCED APOPTOSIS VIA REDUCTION OF ENDOPLASMIC RETICULUM STRESS

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10.1136/heartjnl-2011-300867.161

Aims 4,4'-diisothiocyano-2,2'-disulfonic acid (DIDS), a non-selective chloride channel blocker, has been shown to prevent apoptosis-induced by pathways of death receptor and mitochondria. However, the relationship between DIDS and apoptosis-induced by endoplasmic reticulum stress (ERS) remains undefined. Thus the present study aims to explore whether DIDS attenuates ERS-induced cardiomyocyte apoptosis.

Methods Neonatal rat cardiomyocytes were exposed to Tunicamycin (Tm), an inducer of ERS, in the absence or presence of DIDS. Cell viability, apoptosis and protein expressions of GRP78, CHOP were determined by MTT assay, flow cytometry and western blotting, respectively.

Results Tm (100 ng/ml) resulted in a significant decrease in cell viability ($p < 0.05$). Presence of DIDS (75 $\mu\text{mol/l}$) markedly improved cell viability. Tm also increased cardiomyocyte apoptosis ($p < 0.05$). Exposure of cardiomyocytes to Tm resulted in threefold and 6.5-fold increases of GRP78/BiP and CHOP, respectively ($p < 0.05$). Co-treatment of DIDS and Tm led to not only the decrease of GRP78, but also attenuation of CHOP ($p < 0.05$).

Conclusion These findings demonstrate that DIDS protects cardiomyocytes against Tm-induced apoptosis through attenuation of endoplasmic reticulum stress.