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**Objectives** To study the effects of Wedelolactone (Wed) on the protein expression of apoptosis-associated Bcl-2, Bax and PARP (89KD) in the primary cultured rat cardiomyocytes subjected to anoxia/reoxygenation injury.

**Methods** The primary cultured neonatal rat cardiomyocytes were pretreated with Wed (0.2, 2, and 20 μmol/l) or Wed (2 μmol/l) for 1 h, respectively, and subjected to anoxia for 3 h and subsequently reoxygenation for 2 h. Cell viability, Creatine kinase (CK) and lactate dehydrogenase(LDH) activity in medium were measured. Terminal deoxynucleotidyl transferase d-UTP nick end labelling (TUNEL) staining was performed using an In Situ Cell Death Detection kit on rat cardiomyocytes. The expression of Bcl-2 and the apoptotic protein Bax and PARP (89KD) were detected by Western blotting.

**Results** Compared with that of the control group, the numbers of TUNEL-positive nuclei were significantly increased in cardiomyocytes after 3 h of anoxia and 2 h of reoxygenation. Bcl-2 protein in cardiomyocytes decreased significantly ($p<0.01$) and the expression of Bax protein and PARP (89KD) in cardiomyocytes increased significantly after reoxygenation. ($p<0.01$), Cell viability decreased obviously after anoxia/reoxygenation ($p<0.05$). Compared with that of the anoxia /reoxygenation group, Pretreatment with different concentration Wed decreased LDH activity and increased the survival of the cells significantly ($p<0.05$). The expression of Bcl-2 protein in the Wed (2 μmol/l) groups increased significantly ($p<0.05$) and the expression of Bax protein decreased significantly ($p<0.05$).

**Conclusions** The Wed pre-treatment before ischaemia has antiapoptotic effects on neonatal rats myocardial cells undergoing anoxia/reoxygenation, the underlay mechanism might be attributed to the up-regulated the expression of Bcl-2 gene and the inhibited the expression of Bax and PARP(89KD) gene expression.

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## EFFECTS OF WEDELOLACTONE ON THE PROTEIN EXPRESSION OF APOPTOSIS-ASSOCIATED BCL-2, BAX AND PARP (89KD) IN THE PRIMARY CULTURED RAT CARDIOMYOCYTES SUBJECTED TO ANOXIA/ REOXYGENATION INJURY

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