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ACTIVATION OF RETINOID X RECEPTOR PROTECTS AGAINST HYPOXIA-REOXYGENATION INDUCED APOPTOSIS AND LOSS OF MITOCHONDRIAL MEMBRANE POTENTIAL IN H9C2 RAT VENTRICULAR CELLS

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Objectives Retinoid X receptor (RXR) plays a central role in the regulation of intracellular receptor signalling. To determine the effect of activating RXR on rat cardiomyocytes apoptosis and the loss of mitochondrial membrane potential in hypoxia-reoxygenation induced oxidative injury.

Methods The model of hypoxia/reoxygenation (H/R) injury was established through hypoxia for 6 h and reoxygenation for 4 h in cardiomyocytes of H9c2. We measured the survival rate by Typan blue exclusion, apoptosis rate of cardiomyocytes by FACS analysis, and mitochondrial membrane potential by JC-1 fluorescent probe. All measurement data were expressed as mean $\pm$ SD of mean, and statistically analysed using one-way ANOVA analysis and Dunnett test. Differences were considered significant when P was <0.05.

Results We showed that the RXR agonist 9-cis retinoid acid (9-c RA) protected cells from H/R-induced cell injury and loss of mitochondrial membrane potential, and the protective effect of 9-c RA against H/R was abolished when pretreated with HX531 (RXR pan-antagonist).

Conclusions Activating RXR inhibited apoptosis and prevented the loss of mitochondrial membrane potential induced by H/R injury in rat cardiomyocytes H9c2, which suggests that stabilisation of mitochondrial membrane potential may be involved in the protection of activating RXR against oxidative stress in H9c2.