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Adiponectin Attenuates Hypoxia/Reoxygenation-Induced Cardiomyocyte Injury Through Inhibition of Endoplasmic Reticulum Stress

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[**KEY WORDS**] Adiponectin; Hypoxia-Reoxygenation; Endoplasmic Reticulum Stress; p38 Mitogen-Activated Protein Kinase; Cell Death

Background and Aim Adiponectin (APN) is a potent cardioprotective molecule. The present study aims to investigate the underlying mechanism(s) for its cardioprotective effect. **Methods** Primary cardiomyocytes were isolated from neonatal rats and an in vitro model of hypoxia-reoxygenation (H/R) was established. The cardiomyocytes were randomly divided into six groups: saline group (control), dithiothreitol (DTT) group (5 mmol/L DTT for 2 h), H/R group, H/R + APN group (incubation with 30 mg/L

APN, followed by H/R), H/R + APN + SB203580 (SB) group (treatment with 30 mg/L APN and 5 μmol/L SB, followed by H/R), and H/R + SB group (exposure to 5 μmol/L SB and then H/R). Cell death was detected by measuring lactate dehydrogenase (LDH) release. The expression levels of hypoxia-inducible factor-1 alpha (HIF-1α) and endoplasmic reticulum (ER) stress-related genes including GRP78, caspase-12, C/EBP homologus protein (CHOP), and p38 mitogen-activated protein kinase (MAPK) were examined. **Results** Cardiomyocytes exposed to H/R showed a significant increase in LDH leakage and HIF-1α protein levels compared with the control cells ($P<0.05$). The H/R-provoked cell death was profoundly attenuated by the pretreatment with APN alone, SB alone, or both, which was coupled with decreased expression of GRP78, caspase-12, CHOP, and p38 MAPK. **Conclusions** These results provide new insights into the mechanism of APN-mediated cardioprotection, which may be partially due to inhibition of ER stress response.

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