



■ SUPPLEMENTARY ONLINE DATA

Angiotensin II and the ERK pathway mediate the induction of myocardin by hypoxia in cultured rat neonatal cardiomyocytes

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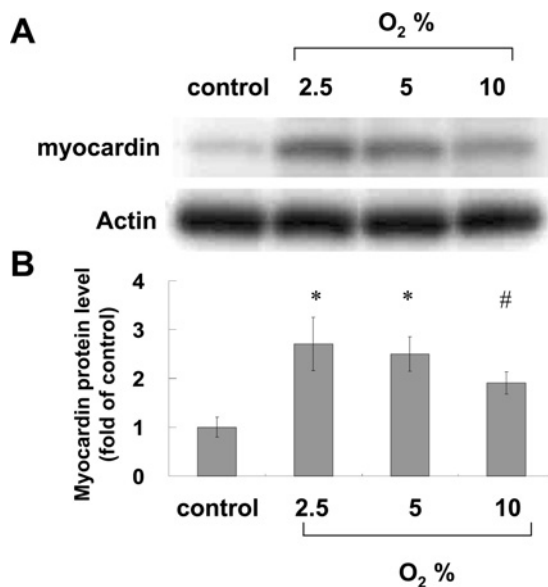


Figure S1 Hypoxia increases the expression of myocardin protein in cultured rat neonatal cardiomyocytes

Neonatal cardiomyocytes were subjected to normoxia or different degrees of hypoxia for 4 h, and total cell lysates were immunoblotted with the anti-myocardin antibody. Actin is used to show equal amounts of protein loading in each lane. #*P* < 0.05 and **P* < 0.01 compared with normoxia control (*n* = 3).

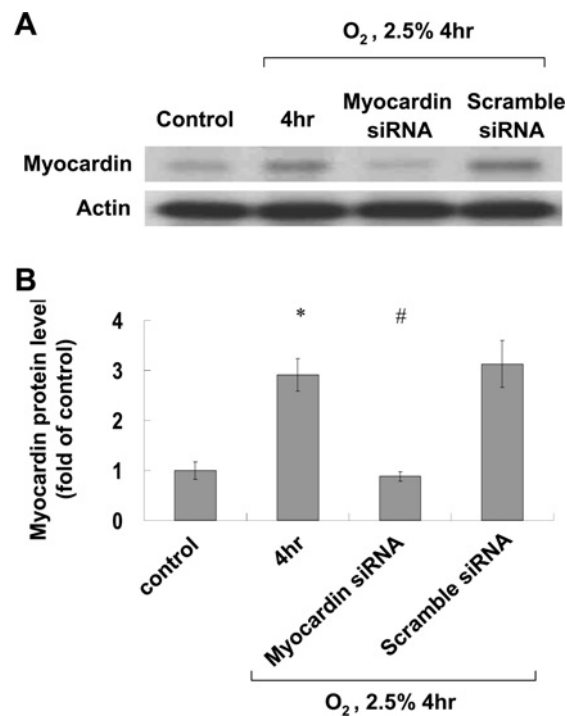


Figure S2 Effect of myocardin siRNA on myocardin expression in cardiomyocytes under 2.5% O₂

Myocardin protein levels increased and reached a peak after hypoxia for 4 h, which was suppressed by myocardin siRNA. Actin is used to show for equal amounts of protein loading in each lane. **P* < 0.01 compared with normoxia control; #*P* < 0.01 compared with 4 h (*n* = 3).

Myocardin confocal

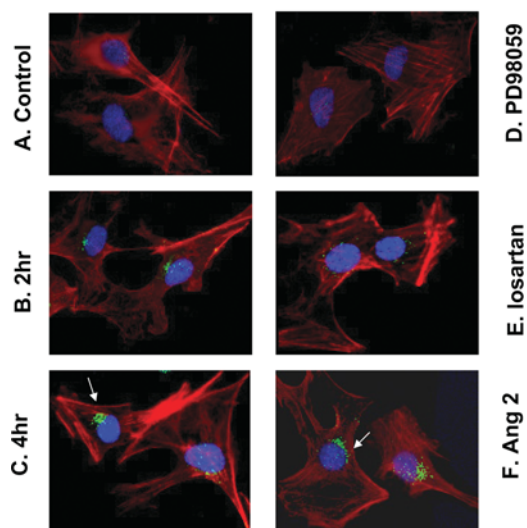


Figure S3 Confocal microscopy identifies the expression of myocardin in the nucleus of neonatal cardiomyocytes after hypoxia

(A) Cardiomyocytes under normoxia. (B and C) Myocardin protein (green) levels increased in the nucleus (blue) of neonatal cardiomyocytes from 2 to 4 h. (D and E) ERK pathway inhibitor (PD98059) and ARB (losartan) suppressed the expression of myocardin. (F) AngII increased the expression of myocardin in the nuclei of neonatal cardiomyocytes.

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