ABSTRACT

CHRONIC INTERMITTENT HYPOXIA PROMOTES DIFFERENTIAL EXPRESSION OF STRESS PROTEINS FOLLOWING *in vitro* HYPOXIA IN GUINEA-PIG HEARTS

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The rapid induction of heat shock proteins (eg. Hsp70 and Hsp90) occurs in response to heat, ischaemia and hypoxia. An increase in these proteins has been implicated in myocardial protection (Yellon and Latchman, *J. Mol Cell Cardiol* 1992, 24:113-124). We tested the hypothesis that 21 days chronic intermittent hypoxia training (IHT) alters the expression of Hsp70 and Hsp90 in the guinea pig heart as well as their expression in response to severe *in vitro* hypoxia in the isolated guinea pig atrial preparation. An IHT group was exposed to 8% O_2 /0.3%CO_2 for 12 h /day for 21 days. A normoxic control group (Control) was exposed to room air for 21 days. After the experimental period, both groups were exposed to episodes of acute *in vitro* hypoxia (0,5,10 and 20%O_2 / 5%CO_2 3-4 min. each). In both groups, Hsp70 and Hsp90 protein content were determined by Western blotting using specific monoclonal antisera. IHT did not alter ventricular Hsp70 and HSP90. In atrial samples that were exposed to acute *in vitro* hypoxia, expression of both HSPs were significantly higher in IHT and Control groups. However, in the IHT group, atrial Hsp90 expression was significantly lower than the Control group following acute *in vitro* hypoxia. A similar trend was observed with Hsp70 expression. These results show that IHT training inhibits cardiac HSP expression during acute *in vitro* hypoxia. This suggests that IHT may minimize stress induced responses in the heart.