HEAT SHOCK TREATMENT PRIOR TO DIVE INCREASES SURVIVAL IN RATS

Bye A., Medbye C., Brubakk A.O.
Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Trondheim, Norway.

Background and objectives. Heat shock represents a non-pharmacological preconditioning strategy, which can lead to protection against subsequent cellular strain, for instance diving. Exposure to heat shock involves up-regulated amounts of heat shock proteins, and might be one of the crucial mechanisms behind the protective effect. In this study we focus on the heat induced heat shock protein 70 (HSP70) and heat shock protein 90 (HSP90), in addition to endothelial nitric oxide synthase (eNOS) which is regulated by HSP90. The aim is to explore the possibility that exposure to heat shock 24 hours before a severe dive, could increase the survival in rats.

Materials and methods. 24 Sprague Dawley rats were divided into a heat shock and a control group. The heat shock group was heated for 15 minutes at a core temperature of 42 ± 0.5° C. The following day the rats from both groups did a simulated dry air dive in a hyperbaric chamber (700 kPa at 200 $kPa.min^{-1}$). After 45 minutes the rats were returned to surface pressure at 50 $kPa.min^{-1}$ and anaesthetized. Aorta and left ventricle were isolated, and the tissue was homogenized for Western blot analyses.

Results and discussion. In the heat shock group 6 out of 12 rats survived, while 1 of 12 control rats survived. This result* is significant, and indicates that the prior heat shock induced some important cellular changes which avoid apoptosis or necrosis. Western blot analyses showed an 8.0-fold increased HSP70 content in left ventricle and a 4.4-fold increase in aortic tissue in the heat shock group versus the control group. There was no change in eNOS and HSP90 levels between the two groups. Since the heat shock group had elevated amounts of HSP70, it's a possibility that HSP70 is involved in the observed protective mechanism. HSP90 and eNOS does not respond the same manner, which can be due to a poorer heat induction of HSP90 compared to HSP70. This supports the theoretical findings that HSP90 is less heat induced than HSP70, and that the expression of HSP90 and eNOS is closely related. The results suggest that HSP90 and eNOS are less important in the protective mechanism behind whole body hyperthermia, but further analyses are required to reveal the complete mechanism behind heat shock preconditioning.

Conclusion. Heat shock treatment prior to dive causes elevated levels of HSP70 and increases the survival in rats.

* Previously reported at the 2003 EUBS