Heat Shock Protein 72 rises following a diving stress and this rise is accelerated following preconditioning using TEX-OE.

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Introduction

Diving may subject the body to a number of stresses, which may include cold, hyperoxia and hypoxia. Heat shock protein (HSP) are rapidly expressed by individual cells in response to any adverse environmental stress and serves to protect those same cells from the harmful effect of the stress conditions. In recent reports HSP has been linked to cellular preconditioning and salvage and has been shown to induce ischaemic tolerance in animals as well as antagonizing the oxidative damage due to acute and severe tissue hypoxia.

In humans, the ability to react to stress and aggressive forces becomes less and less pronounced with age. TEX-OE is a plant extract which has been found to have the ability of enhancing HSP synthesis in animals and has been shown to have, by this scientific team, a significant protective effect in animals against the permanent tissue damage produced by a severe decompression insult. It also seems to counteract the effects of aging on the stress response.

Method

The subjects were 12 volunteer fit divers. A baseline blood sample (0.5cc) was taken from each subject using a standard aseptic technique and stored. The divers were asked to carry out a normal sea-water dive to 35 meters for 20 minutes on air. Decompression was carried out using a dive computer. A second blood sample (0.5cc) was taken from each subject at one hour from the onset of the dive and a third sample one hour after the second. All samples were stored in a heparinazed tube at -20 degrees C. Doppler precordial readings were taken between 20 to 40 minutes post surfacing to elicit any venous bubbles (DAN Europe' s Safe Dive protocol). The subjects were followed up for 24 hours post dive, for any signs and symptoms of DCI. None were reported.

The same group of divers repeated exactly the same dive one week later, this time after ingesting 450mg of TEX-OE two hours before their last meal the night before, and they were monitored using the same protocol. Five of the divers were asked to repeat the same dive 24 hours and 48 hours later where they were again monitored using the same protocol. The blood samples were examined for the levels of HSP 72 as determined using ELISA.

The trial was then repeated by subjecting 6 of the divers to the same dive profile but as a dry simulated dive in a hyperbaric chamber in order to show that a dry chamber dive will produce a similar result to an actual in water dive and to further define the time and rate of rise of HSP through serial blood sampling.

The 6 recreational divers, who had ingested TEX-OE capsules 12 hours previously, were monitored before, during and after the simulated recreational dive for HSP production again by means of venous blood sampling taken pre-dive, on reaching bottom, at 15 minutes, at the decompression stop, at 30 minutes, 60 minutes and 90 minutes post dive. Analysis of the samples was again carried out using an ELISA method.

Results

Table 1a shows that at one hour after the start of the first sea water dive there was minimal or no rise in the level of HSP recorded while all divers showed a rise at the two hour reading. Table 1b shows the readings obtained following the second sea water dive one week later. The HSP level can be seen to rise significantly within one hour of the start of the dive and reached maximal levels in two hours. This effect was noted in the 24hours and 48hour dives.

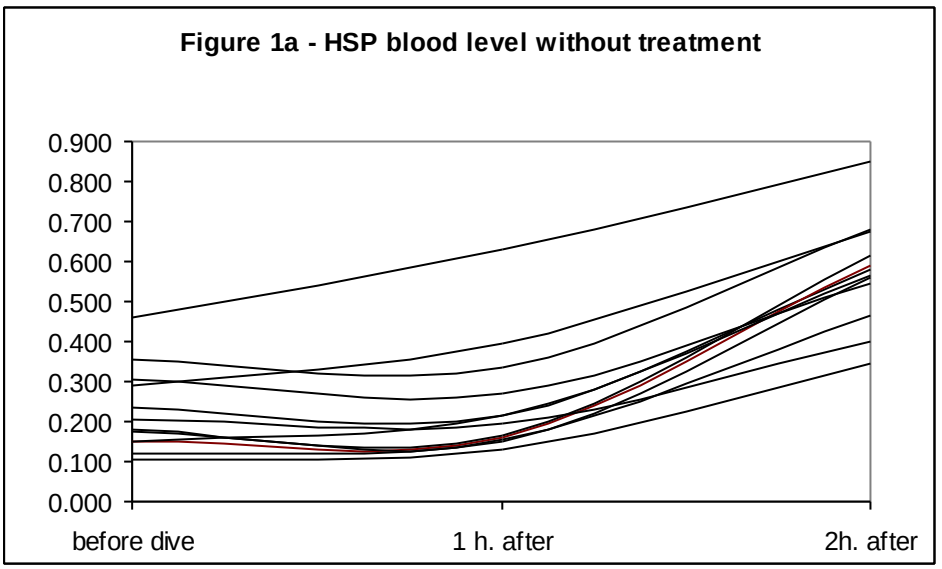
Table 1a - No treatment

	before dive	1 h. after	2h. after
JM	0.355	0.335	0.679
GM	0.235	0.215	0.582
SF	0.290	0.397	0.677
CC	0.462	0.629	0.851
GG	0.105	0.128	0.346
CV	0.151	0.160	0.591
EC	0.121	0.154	0.467
JC	0.177	0.164	0.614
CS	0.181	0.151	0.562
DL	0.148	0.215	0.565
JJ	0.303	0.268	0.544
SD	0.203	0.194	0.4

Table 1 b - TEX-OE treatment

	Before dive	1 h. after	2h. after
JM	0.267	0.502	1.878
GM	0.299	1.287	4.300
SF	0.309	0.535	1.728
CC	0.211	1.167	5.542
GG	0.039	0.527	0.461
CV	1.178	1.343	1.140
EC	0.308	0.781	2.539
JC	0.134	0.501	0.567
CS	0.129	0.559	0.497
DL	0.261	0.543	0.520
JJ	0.314	0.566	0.560
SD	0.247	0.527	0.569

Figures 1a and 1b represent these readings graphically. One diver started the dive with a relatively high baseline and there was no significant change recorded at the one and two hour readings.



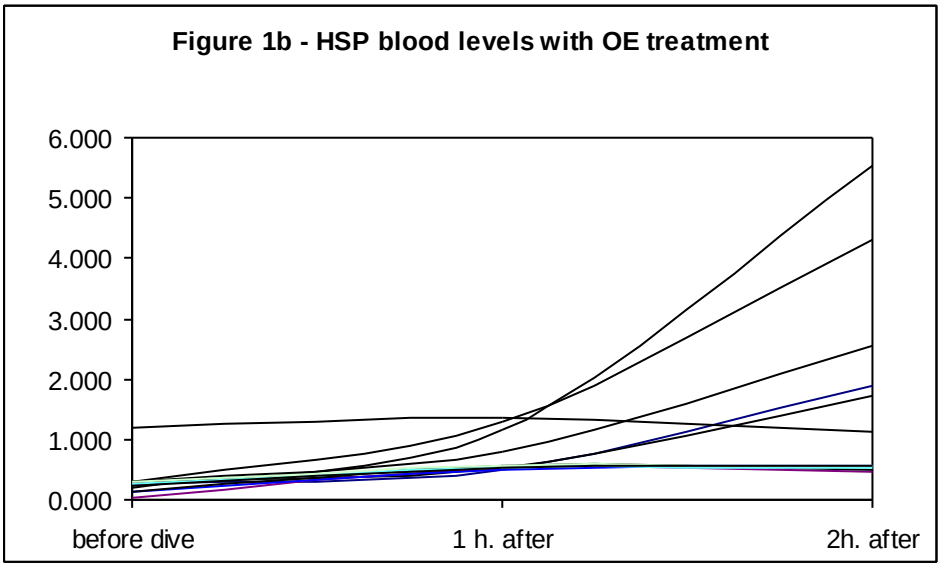
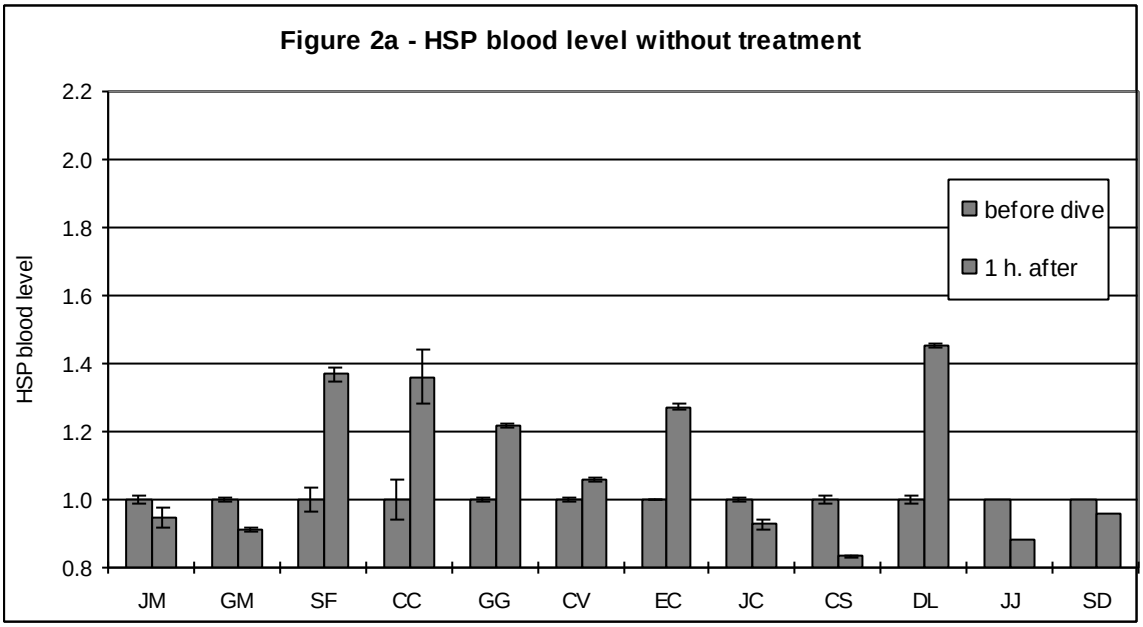


Figure 2a and 2b demonstrate the same results when the base line HSP readings have been adjusted in all the subjects to a standard reference point.



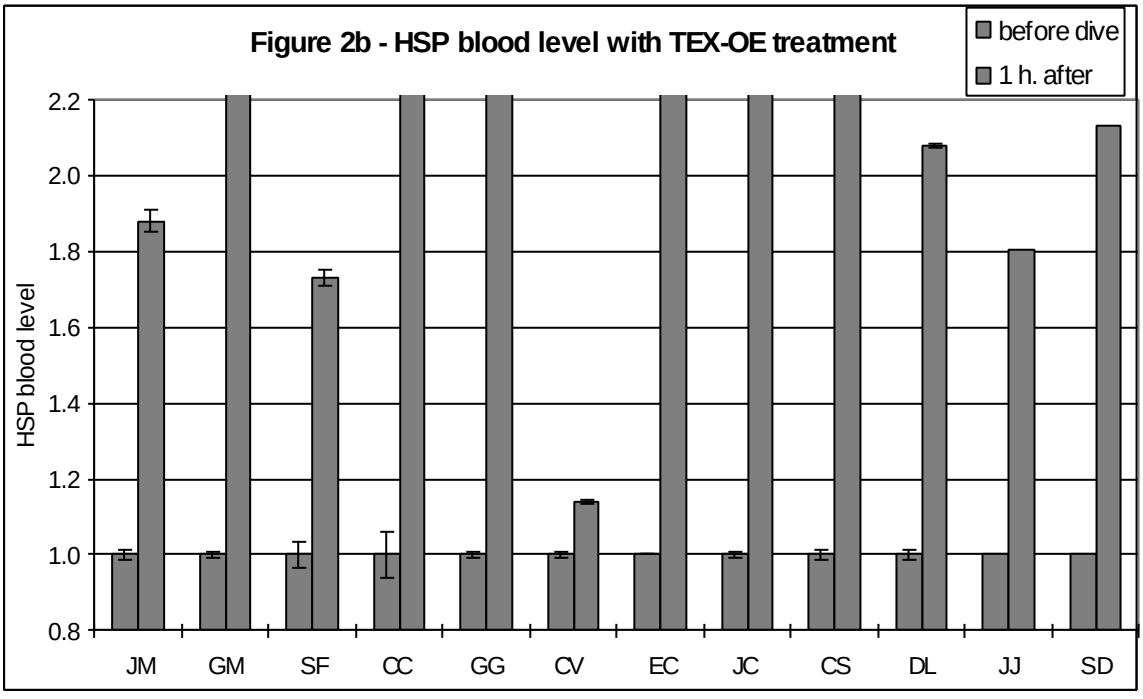


Figure 3 shows the results of the chamber dive. A significant rise in the level of HSP 72 can be seen to occur within fifteen minutes of the onset of the dive with a maximal rise within 30 minutes.

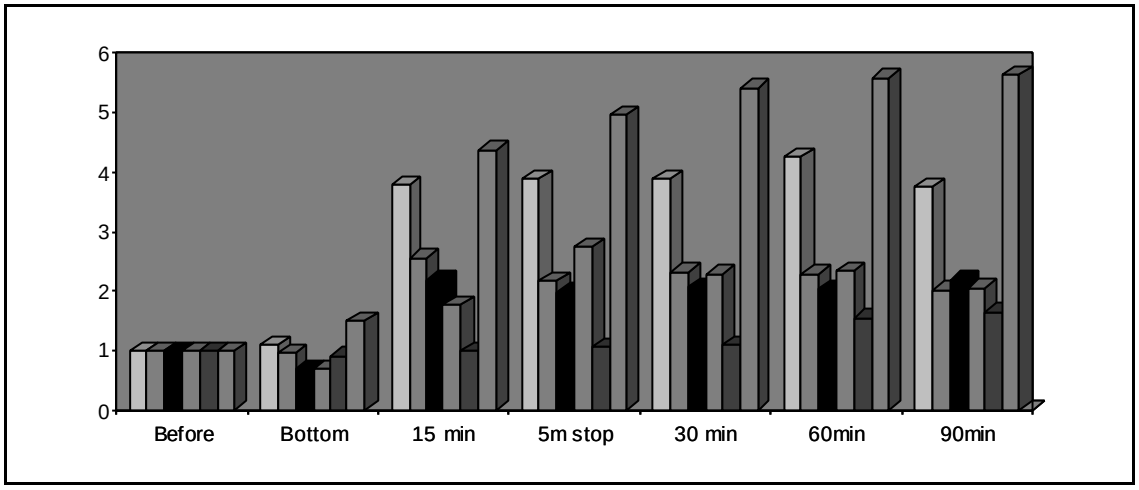


Figure 3

The diver who had a low response during the in water dive again had a similar response during the dry dive. it is likely that his particular response may be related to his occupation which is that of a farmer and so is continually exposed to numerous naturally preconditioning stresses such as the sun and the elements. It was also noted that HSP production following preconditioning is not influenced by the age of the subject.

Conclusion

Diving activities seem to produce a type of physical stress capable of triggering the intracellular production of heat stress proteins. At the present time it is uncertain which elements of the dive are actually involved. The protective effect of a number of these proteins has been repeatedly documented in a number of publications and the possibility that a higher level of these proteins will enable the individual cell to resist the damage produced by Decompression Illness has to be investigated further. However our preliminary animal and human studies have shown that there may be a clear role for preconditioning and in particular TEX-OE as a prophylactic protective agent in diving. It is intended to carry out an extensive multi-centric study looking into this effect and to validate our hypothesis that early high levels of HSP as induced by TEX-OE exert a protective effect against the physical and physiological stresses inherent to diving and to the tissue damage following DCI

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Titolo: Diving causes a rise in the level of Heat Shock Protein (HSP) and early high levels of HSP are induced following pre-dive ingestion of TEX-OE
Oggetto:
Autore: Dr. Cali' Corleo
Parole chiave:
Commenti:
Data creazione: 27/10/99 11.55
Numero revisione: 2
Data ultimo salvataggio: 27/10/99 11.55
Autore ultimo salvataggio: Alessandro Marroni
Tempo totale modifica1 minuto
Data ultima stampa: 11/12/01 10.13
Come da ultima stampa completa
Numero pagine: 6
Numero parole: 1.173 (circa)
Numero caratteri: 6.691 (circa)