

Decompression injury and cellular defense mechanisms

PhD student Lise Fismen

Supervisors:

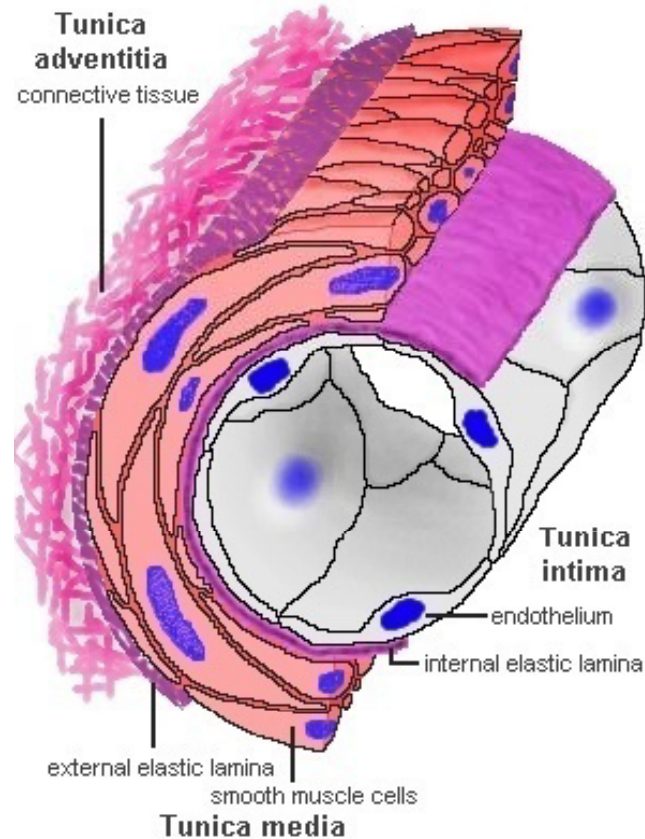
professor Asbjørn Svardal, UiB
dr. philos Rune Djurhuus, NUI AS



Preventing bubbles and DCS

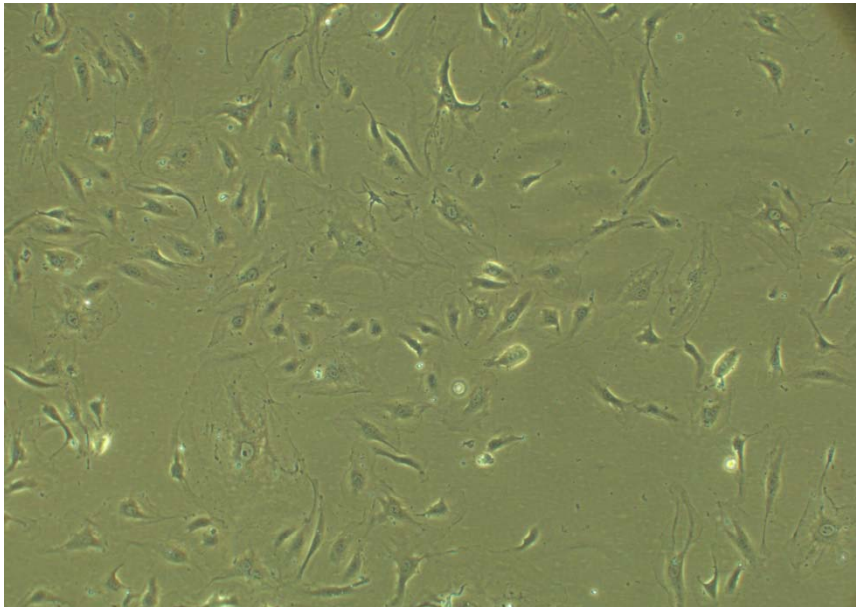
- Heavy exercise 24 hrs prior to diving prevents bubble formation in animals and humans
- A heat stress 24 hrs prior to diving protects rats against decompression sickness (DCS), no effect on bubble formation
- Protection against DCS associated with production of stress proteins

The endothelium is a prime target for damage caused by bubbles

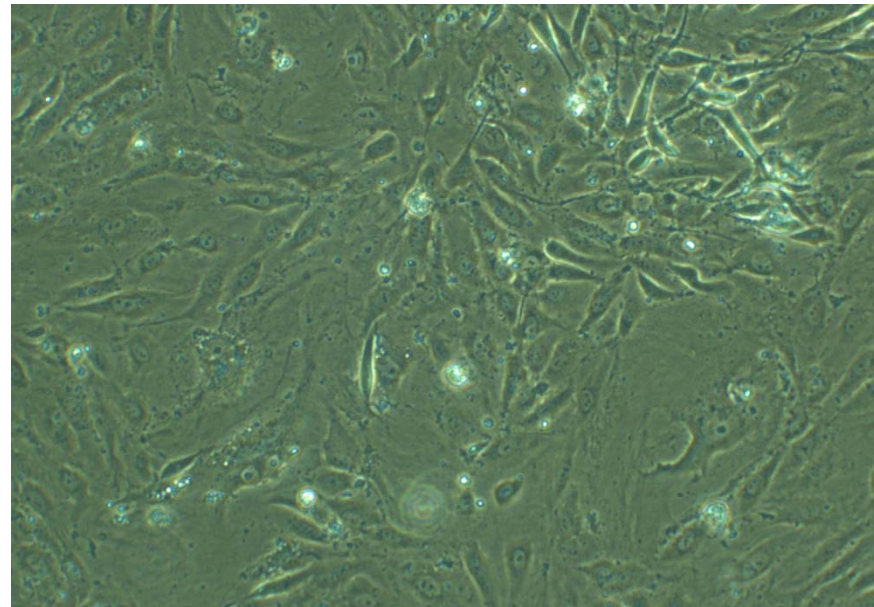


<http://www.lab.anhb.uwa.edu.au/mb140/CorePages/Vascular/Images/VesWall.jpg>

Human blood vessel (endothelial) cells as a model system



Exponential growth phase



Confluent phase

AIM of project

To study the effect of


heat stress and simulated diving on

biochemical factors in blood vessel cells

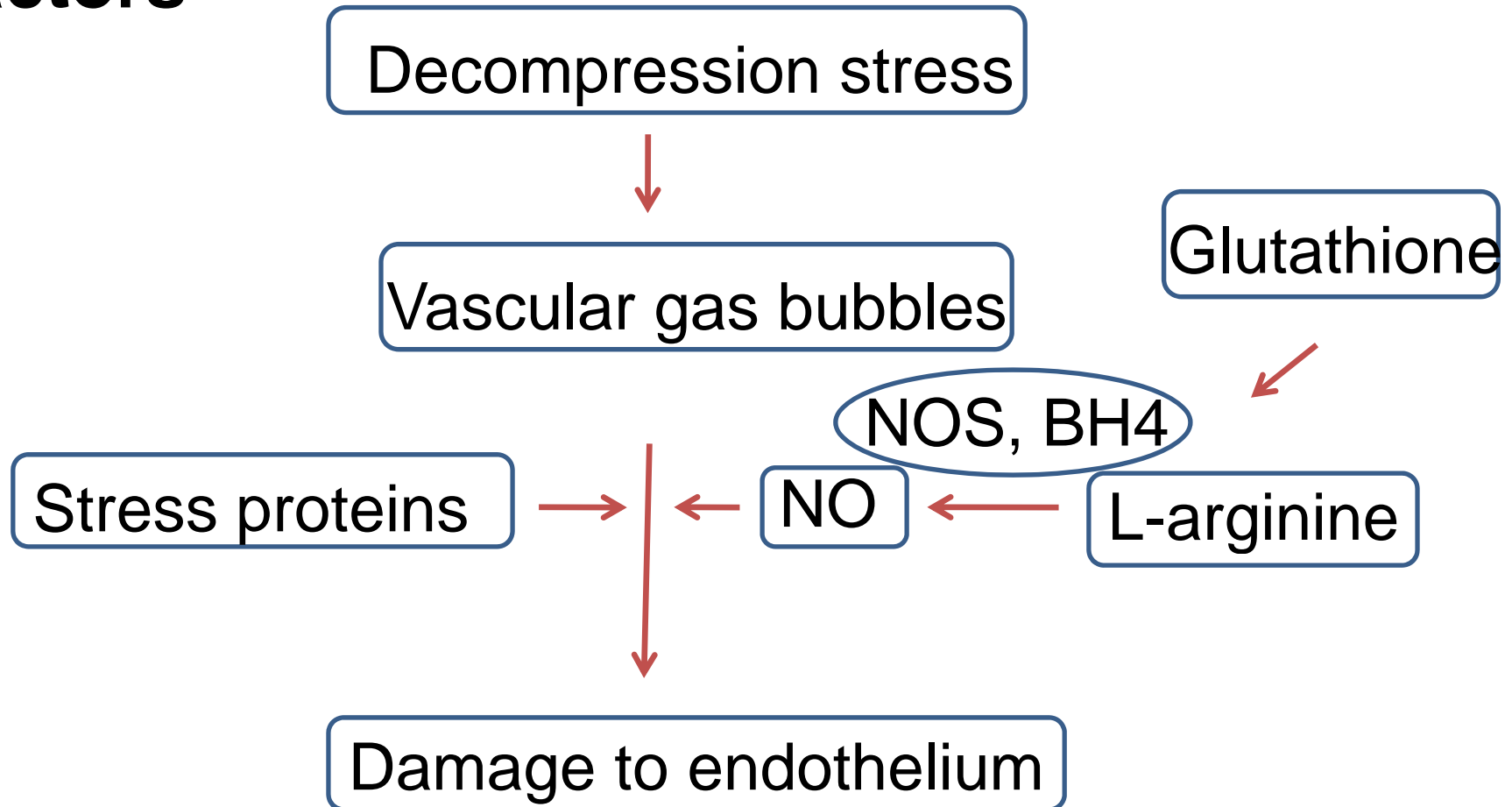
that can protect against damage due to

decompression

Biochemical factors of interest

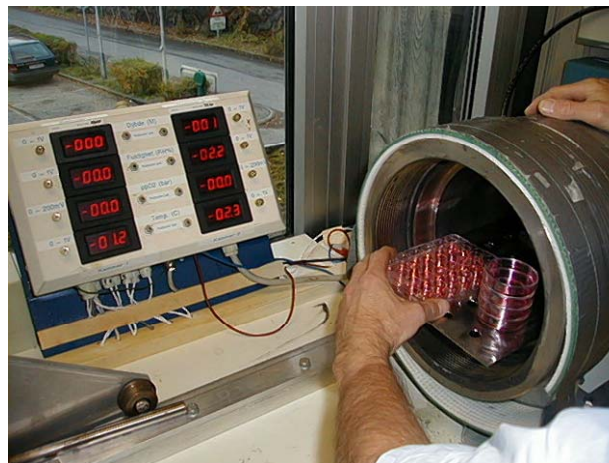
- Glutathione (GSH) – major cellular antioxidant
- Nitric oxide (NO) –  signalling molecule mediating vasodilation, synthesised by nitric oxide synthase (NOS)
- Tetrahydrobiopterin (BH₄) – important cofactor for NOS - dependent on redox status

Why we are interested in these biochemical factors

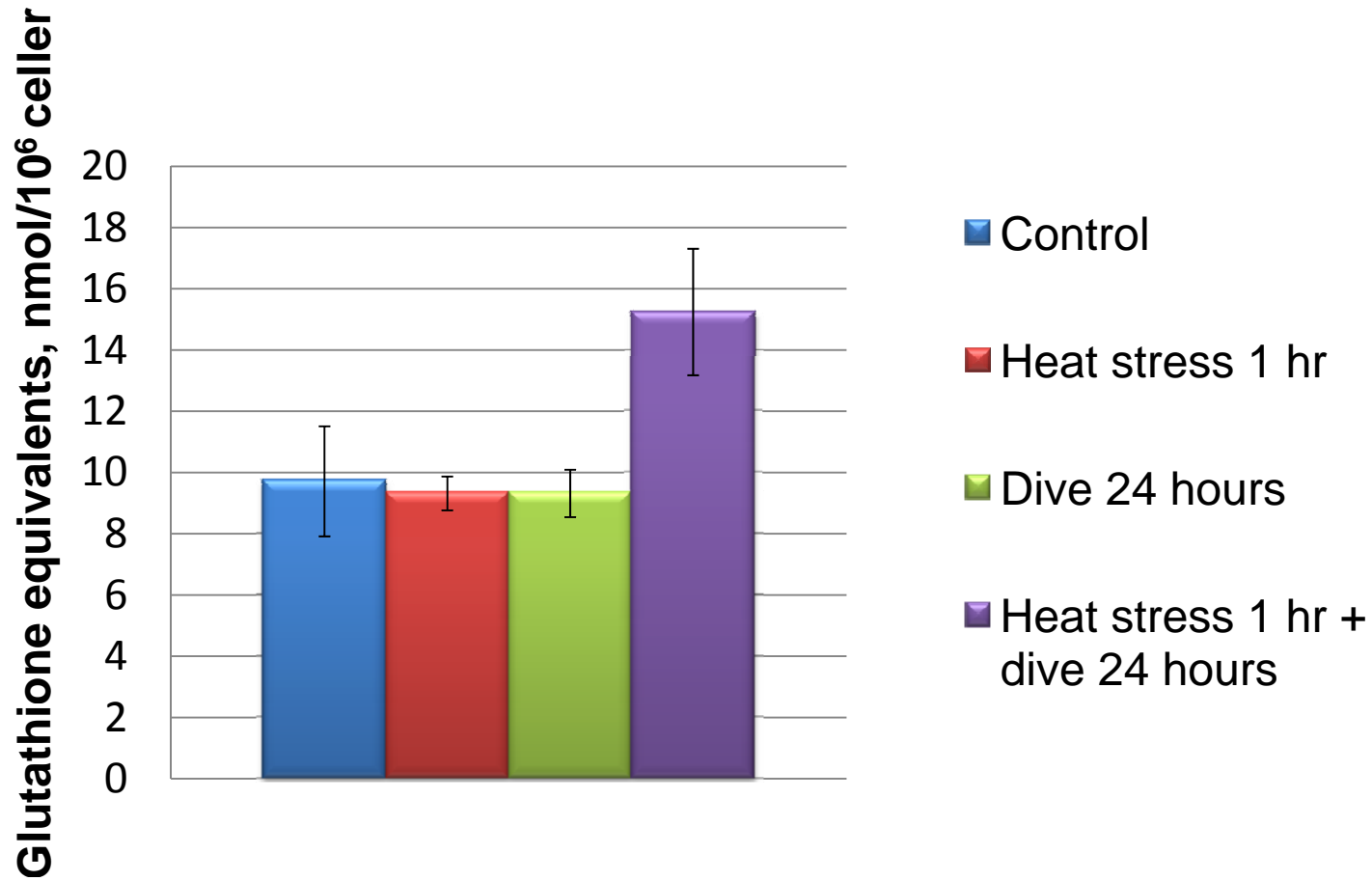


Simulated dive in pressure chamber

Exposure group	0	Hours	24	48
Control	Std. conditions			
HS	HS 45 ⁰ C, 1 h	Std. conditions		
Dive	Std. conditions		Dive 24 h	
HS+dive	HS 45 ⁰ C, 1 h	Std. conditions	Dive 24 h	



Total glutathione in cell extracts



Why is this useful?

Knowledge about biochemical factors may

- explain the mechanisms resulting in decompression damage
- be utilised to prevent damage
- be of importance in future diving procedures and divers' health

