Can research on the role of NO in hypoxia tolerance tell us something useful about critical illness?

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One in four of us will end up on Intensive Care at some stage of our life, and a third of those will not leave the ward alive. Cellular hypoxia, with systemic inflammation, is a near-ubiquitous challenge in this setting. Current biomarker approaches focus on early detection of tissue damage, with supplementary O₂ administered frequently to treat hypoxia. Hypoxic signalling, innate immunity and inflammation are under tight redox control; the production of nitric oxide (NO) and reactive oxygen species (ROS) is fundamental to “redox signalling” and thus O₂ utilisation, cellular energy production and inflammation. While these processes are relatively well characterised in cellular systems, their relevance for human physiology is less well understood. This is in part due to the difficulties associated with carrying out controlled clinical studies in the critical care setting.

In my talk I will present preliminary results from an alternative experimental approach that employs a combination of whole-body physiology and multi-biomarker research focussing on the NO/oxidative stress/redox signalling pathway during exposure of healthy human subjects to increasing levels of hypoxia. The data obtained so far suggest that an adequate production of NO is crucial for the ability of humans to tolerate a reduction in O₂ availability. Thus, NO enhancing strategies rather than O₂ supplementation may improve survival from conditions associated with reduced O₂ availability.

Thursday 8th November at 15:15
Room 127 (zoofys kaffestue), building 1131