zoophysiology seminar at Aarhus University

# Oxygen Sensing or Just Passing Gas: $H_2S$ as the Mediator of Hypoxic Signaling


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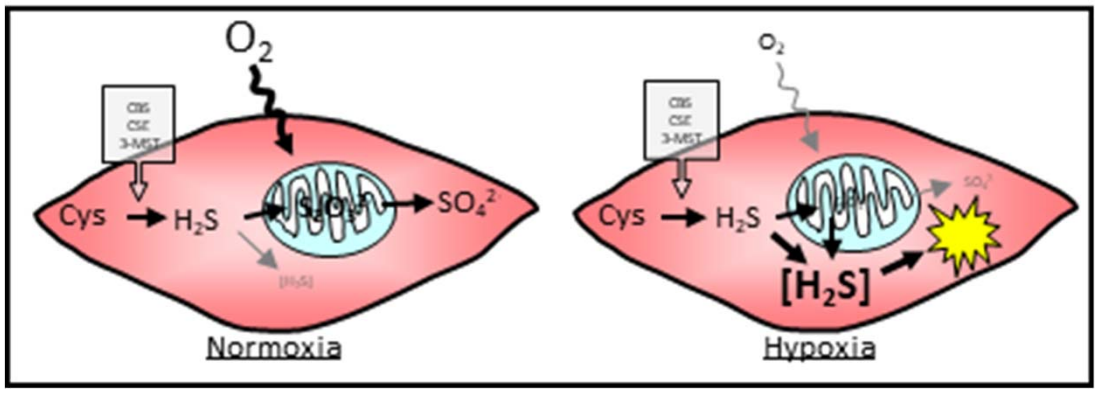
Hypoxia is an omnipresent threat to aerobic organisms and its detection is implicit for survival. While responses to hypoxia are well characterized, the identity of the oxygen "sensor" is less clear. In 2006 we proposed that metabolism of hydrogen sulfide ($H_2S$) was a rapidly responding $O_2$ sensing mechanism in which constitutively produced $H_2S$ is rapidly oxidized during normoxia, whereas oxidation fails during hypoxia and the ensuing rise in intracellular $H_2S$ initiates appropriate effector responses. This hypothesis is supported by observations that; 1) hypoxia and $H_2S$ produce identical responses in a wide variety of physiological systems, 2) compounds that stimulate or inhibit $H_2S$ production stimulate or inhibit hypoxic responses, 3) failure of $H_2S$ oxidation occurs at physiologically relevant $P_{O_2}$s, and 4) this process occurs in mitochondria, the presumptive $O_2$-sensing organelles. Recent work will also be discussed showing that $H_2S$ metabolism mediates chronic $O_2$ sensing and ischemic preconditioning.


**Thursday May 2nd at 10.30**

*Seminar room at section for Zoophysiology*