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I E C B

HIF and PHDs-mediated hypoxia tolerance.

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Maintaining oxygen (O_2) homeostasis is essential for most organisms and O_2 deficiency (hypoxia), even transient, can produce irreversible damage. Adaptation to reduced oxygen availability is indeed a major physiologic challenge. This adaptation is triggered by the Hypoxia-signalling cascade, which activation is essential during embryonic development and in adulthood but it is also associated with pathologies such as ischemic diseases, inflammatory and metabolic disorders, Alzheimer and cancer.
By driving the primary transcriptional response, the Hypoxia Inducible Factor (HIF) is a key player of the hypoxia-signalling cascade. HIF is mainly regulated through the Prolyl Hydroxylase Domain containing proteins (PHDs). These enzymes trigger the O_2-dependent HIF prolyl- hydroxylation that control HIF stability.
Here, we will discuss our more recent data regarding the study of the molecular mechanisms underlying HIF and PHDs regulation, their implication in physiopathology and the different therapeutic strategies developed so far to target the hypoxia-signalling pathway.

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