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Regulation of gene expression by hypoxia

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Decreased oxygen availability (hypoxia) is a hallmark feature of the microenvironment in a number of conditions including arthritis and inflammatory bowel disease (IBD). Recent advances in our understanding of oxygen-dependent cell signaling have uncovered several mechanisms by which hypoxia impacts upon the development of inflammation through the coordinated expression of adaptive, inflammatory and apoptotic genes. Two central transcription factors involved in the regulation of this response are Hypoxia Inducible Factor (HIF) and Nuclear Factor-κB (NF-κB) which display different degrees of sensitivity to activation during hypoxia. Furthermore, HIF and NF-κB demonstrate an intimate interdependence at several mechanistic levels. Recent studies indicate that these pathways may represent important new therapeutic targets in diseases characterized by hypoxic inflammation.

Where applicable, the authors confirm that the experiments described here conform with The Physiological Society ethical requirements.