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HYPOXIA-INDUCIBLE FACTOR: MODERN VIEWS

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Oxygen is needed by cells to produce enough ATP for metabolic activity. Hypoxia, or oxygen starvation, occurs in human tissues and cells due to a variety of conditions, including heart and lung disease, anemia, and circulatory problems. Depending on the severity, irreversible damage to tissues and cells can occur.

However, hypoxia can also play an important and beneficial role in human physiology and development. It is an essential part of proper embryonic development. Although the exact mechanisms are not known, oxygen tension is associated with neural tube closure, mediation of apoptosis, and proper morphological development during pregnancy. In addition to genetic signals, environmental conditions such as hypoxia have been shown to serve as signals for embryonic development.

Many organisms have developed mechanisms for adaptation to hypoxic conditions. Changes in oxygen levels can lead to the activation or repression of certain homeostatic regulatory genes, allowing tissues and cells to survive despite

fluctuations in environmental conditions. Genes such as HIF-1, which are activated under hypoxic conditions, can interact with enzymes and other transcription factors to control vascularization and tissue growth. While the microenvironment surrounding cancerous tumors is extremely hypoxic, the spread of such masses is often made possible by the activation of HIF-1, which leads to an increase in angiogenesis and thus an increase in oxygen supply to this area.

Given its important function, the manipulation of HIF-1 activity in areas of ischemia and tumor masses has become a major focus of efforts to develop non-invasive, pharmaceutical treatment options for patients with cancer and heart disease. Although no such human protein has been successfully regulated by scientific methods, control of HIF-1 activity is becoming increasingly feasible as the details of its structure, function, and genetic pathway are elucidated.

HIF-1 is a heterodimeric transcription factor consisting of a constitutively expressed β-subunit and an oxygen-regulated α-subunit. Both HIF-1α and HIF-1β proteins contain basic helix-loop-helix motifs that bind DNA and cause subunit dimerization. Both subunits also have a Per-ARNT-Sim (PAS) domain with similar functions. The α-subunit has an oxygen-dependent degradation domain (ODD) that is hydroxylated by proline hydroxylase-2 (PHD-2), making the α-subunit vulnerable to proteasomal degradation under normoxic cellular conditions.

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LIPID PEROXIDATION AS THE MAIN PATHOGENETIC LINK IN THE DEVELOPMENT OF LIVER FAILURE

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Lipids are classically divided into two groups: apolar and polar. Triglycerides (apolar) are stored in various cells, but especially in adipose tissue, they are usually the main form of energy storage in mammals. Polar lipids are structural components of cell membranes, where they are involved in the formation of a cell permeability barrier and subcellular organelles in the form of a lipid bilayer. The main lipid type that defines this bilayer in almost all membranes is phospholipid-based glycerol [1, p. 390]. The importance of the membrane on the physical (phase) state of lipids is evidenced by the fact that lipids can control the physiological state of the membrane organelle by changing its biophysical aspects, such as polarity and permeability. Lipids also play a key role in biology as signaling molecules.

The main enzymes that generate mediators of lipid signaling are lipoxygenase, which mediates hydroperoxyeicosatetraenoic acids (HPETE), lipoxins, leukotrienes, or hepoxylin biosynthesis after oxidation of arachidonic acid (AA), cyclooxygenase, which produces prostaglandins and cytochrome P-450 (CYP), which forms epoxyeicosatriene acids, leukotoxins, thromboxane or prostacyclin. Lipid signaling can occur through the activation of various receptors, including G-protein coupled and nuclear receptors. Members of several different lipid categories have been identified as potent intracellular signaling molecules. Examples of signal lipids include two derived from phosphatidylinositol, phosphates, diacylglycerol (DAG) and inositol phosphate . DAG is a physiological activator of protein kinase C and transcription factor of nuclear factor-kB (NF-B), which promote cell survival and proliferation. Diacylglycerol also interacts