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HIF-1 REGULATES THE TRANSITION TO ANAEROBIC METABOLISM

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HIF-1 may also regulate anaerobic metabolism. When oxygen is available, most cells produce ATP through oxidative phosphorylation. However, in a hypoxic environment, there is a shift towards anaerobic metabolism for cellular energy production. HIF-1 is one of the main genes coordinating this shift by inducing various glycolytic enzymes and glucose transporters such as aldolase A and pyruvate kinase M, which help cells efficiently generate energy in a hypoxic environment. In addition to increasing the expression of these enzymes, HIF-1 reduces mitochondrial oxygen consumption by activating pyruvate dehydrogenase kinase I and stopping the citric acid cycle.

Cancer, inflammation and hypoxia. The environment surrounding metastasizing tumor masses is often hypoxic. HIF-1 is the most important protein

in such masses; it enables tumor progression by inducing alternative metabolic pathways in cancer cells, as discussed above in the context of physiological hypoxia.

The spread of the tumor. Due to its role in hypoxia, HIF-1 plays a critical role in tumor proliferation. As the tumor develops and grows, a hypoxic environment is created due to the extreme energy needs of numerous rapidly dividing cells. Such cell masses often induce angiogenesis to meet the demand for increased oxygen, energy and blood supply. Simultaneously, HIF-1 promotes the transition to anaerobic metabolism. The importance of this transcription factor in tumor cell survival is reflected in the finding that HIF-1α levels in glioma tumor cells increase in proportion to tumor grade.

The mechanisms of tumor survival mediated by HIF-1 are partly disclosed by Semenza et al. on VHL-deficient renal carcinoma cells. HIF-1 has been found to reduce oxygen consumption in these cells by inhibiting C-MYC, a transcription factor that regulates mitochondrial mass and oxygen consumption and is known to be downregulated in various human cancers. Semenza et al. report that HIF-1 reduces C-MYC levels by increasing the transcription of MXI1, a C-MYC repressor, and by increasing the rate of proteasomal degradation of the C-MYC protein. It has been found that reduced levels of C-MYC in these cancer cells ultimately lead to increased glycolysis and reduced mitochondrial respiration, critical characteristics of cancer cells that survive and proliferate in the hypoxic conditions of the tumor microenvironment.

Overexpression of HIF-1 induces apoptosis. Currently, there are many studies on the role of HIF-1 in hypoxia-induced apoptosis of various cell types. For example, Krick et al. recently reported that overexpression of HIF-1 in alveolar epithelial cells leads to increased apoptosis. Although the exact pathways and mechanisms involved in this process remain unclear, evidence suggests that the p53 tumor suppressor is activated under hypoxic conditions. Through interaction with the HIF-1 protein, p53 stabilizes and starts to activate genes such as p21, which in turn cause cell death.

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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