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EXPRESSION AND REGULATION OF THIOREDOXIN IN CANCER CELLS DURING HYPOXIA

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Redox homeostasis is crucial for cell survival. Too much oxygen in the cell leads to oxidative stress through the production of ROS, which reacts with the cells macromolecules causing cell damage and finally cell death. The cells defend themselves against oxidative stress through the production of antioxidants, which either neutralise ROS or reverse ROS-induced damage. In contrast, low oxygen levels in the cell lead to hypoxia. Under hypoxic conditions a signalling pathway involving a key regulator termed hypoxia-inducible factor (HIF) is switched on. HIF drives the induction of many genes controlling multiple cell functions such as angiogenesis, metabolism and apoptosis/survival. Thus, the level of oxygen in a cell dictates the molecular response of cells through modulation of gene expression. Furthermore, both oxidative stress and hypoxia are common features of solid tumours. Both oxidative stress and hypoxia lead to changes in the cellular redox balance within cancer cells. High levels of antioxidants and redox control systems, especially the Thioredoxin system, are often observed in cancer cells and are believed to play a major role in cancer progression. We are currently investigating the expression and regulation of Thioredoxin in breast cancer cell lines cultured in hypoxic conditions and after re-oxygenation.