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Cr(VI)-induced malignant transformation of a bronchial epithelial cell line association with altered mitochondria and bioenergetic phenotypes

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Mitochondria play important roles in cellular energy metabolism, free radical generation, cell signaling and apoptosis. Defects in mitochondrial function have long been suspected to contribute to the development and progression of cancer. Warburg's pioneering work hypothesized that a key event in carcinogenesis involved changes in metabolism, with malignant cells satisfying their energy needs by producing a large portion of their ATP through glycolytic mechanisms, rather than through oxidative phosphorylation. Certain malignant cells have also been reported to have alterations in mitochondrial content as compared to normal cells of the same tissue. In lung fibroblasts, taken from a lung epidermoid carcinoma, low mitochondria content was associated with decreased oxidative phosphorylation and increased glycolysis. Also, recently, it was revealed that, *in vitro* Cr(VI)-induced malignant transformation of BEAS-2B cells was associated with the inhibition of mitochondrial pathway of apoptosis.

Aiming to establish whether the malignant transformation of bronchial epithelial cells was paralleled by changes in cellular bioenergetic and mitochondrial phenotypes, we evaluated the energy metabolism, the mitochondria membrane potential and the mitochondria content in a normal bronchial epithelial cell line and in its malignant derivatives. To this end, the mitochondria membrane potential was evaluated by flow cytometry using the JC-1 fluorescent probe. Fluorescence microscopy was used to evaluate the mitocondria morphology and number, and ^{1}H spectroscopy was used to assess the cell's bioenergetic phenotype. Our results revealed that the more malignant phenotypes correlate with increased mitochondria biogenesis, decreased membrane potential and altered bioenergetic phenotype.

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