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Metabolic changes in human bronchial epithelial cells upon chronic exposure to hexavalent chromium

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Some hexavalent chromium [Cr(VI)] compounds are well established occupational respiratory tract carcinogens. However, despite a very large number of studies, the mechanisms of Cr(VI)-induced malignization at the cellular and molecular levels are only now beginning to be understood with more detail. It has been known for decades, since the seminal studies of Otto Warburg in the 1920s, that most solid tumors exhibit a specific metabolic pattern, characterized by a strong contribution of lactic fermentation to the overall ATP production, even in the presence of ample oxygen. This particular metabolic reprogramming, known as the Warburg effect, provides the background for several diagnosis and therapeutic approaches, such as PET (positron emission tomography) and the design of inhibitors of glycolytic enzymes, respectively. Nevertheless, the exact role of the Warburg effect in carcinogenesis and, in particular, in Cr(VI)-induced lung cancer, remains elusive. In this study, the gradual changes in energy metabolism occurring during the chronic exposure of human bronchial epithelial cells, the main targets of Cr(VI)-induced carcinogenicity, to subcytotoxic or mildly cytotoxic concentrations of Cr(VI) were assessed using markers of bioenergetic status, such as glucose uptake, lactate production and adenylate energy charge. Significant changes were observed in all parameters, in a time- and dose-dependent manner, compatible with a role of the energy metabolism in the Cr(VI)-induced malignization process.

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