

Title of the project: Mechanisms of antiproliferative and cytotoxic effects of sodium selenite in colorectal carcinoma cells with differing p53 status.

Grant Agency: Charles University

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Principal Investigator: V. Králová

Co-investigators: E. Rudolf

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Summary of 2009 results

Title of the presentation: Effects of sodium selenite on colorectal carcinoma cells with different p53 genotype.

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Anticancer effects of selenium compounds have been subject of research at all levels from large epidemiological and clinical trials to animal studies and models in vitro. However, the molecular mechanisms that mediate the effect of selenium remain still unexplored. The aim of this project is to study the mechanisms of the effect of sodium selenite in colorectal carcinoma cell lines with different p53 genotype. Model cell lines include the p53 wild type HCT 116 colorectal carcinoma cells and the HCT 116 p53KO cells derived from the original cell line by gene knock-out. During the first year of the research project we tested the effect of sodium selenite on proliferation, metabolic activity, membrane integrity, DNA synthesis and morphology of model cell lines using Brilliant blue total protein assay, WST-1 metabolic assay, Neutral red assay, BrDU incorporation assay and time-lapse videomicroscopy. We showed that sodium selenite inhibits proliferation, metabolic activity and DNA synthesis in both HCT 116 and HCT 116 p53KO cells in a time and concentration dependent manner during 24 h and 48 h time intervals, affects membrane integrity, cellular morphology and causes cell death. Though individual methods differed in their sensitivity, we showed that the p53 wild type HCT 116 cells were more sensitive to sodium selenite treatment than the HCT 116 p53KO cells.

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