THE INFLUENCE OF A HIGH INTAKE OF EXOGENOUS ANTIOXIDANTS ON THE ACTIVITY OF BETA-GLUCURONIDASE (β-GL) IN THE MOUSE LIVER, KIDNEY AND BLOOD PLASMA

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The lysosomal system, which eliminates foreign and endogenous molecules under physiological conditions, is the most developed degradation system in the cell. Lysosomes seem especially sensitive to oxidative stress. β-Glucuronidase is an acid lysosomal glycoprotein in mammalian tissues. It displays a unique subcellular distribution. This enzyme is included in the detoxification and elimination of hydrophobic xenobiotic compounds via the conjugation of hydroxyl or other reactive groups to form polar compound, such as β-D-glucuronides. The interactions of antioxidants and the activity of lysosomal hydrolases in connection with these reactions in subcellular fractions are unknown. This study aims to analyse the influence of high doses of exogenous antioxidants – reduced glutathione (GSH, 100 μg/g b.w.; vitamin C (L-ascorbic acid, 250 μg/g b.w.); vitamin A (Vitaminum A, 1.5 μg/g b.w.) and vitamin E (Tocopherolum aceticum, 10 μg/g b.w.) – on the reactivity of beta-glucuronidase (β-GL) in the blood plasma and subfractions (cytosol, microsomal, lysosomal) of mouse hepatocytes and kidney cells.

All the biochemical assays were determined spectrophotometrically using a Lambda Bio 20/1998 spectrophotometer (Perkin Elmer). The differences between the control and experimental groups in every fraction were analysed via a three-way analysis of variance, using the Kramer’s test in SAS software package.

Our studies showed the high activity of β-GL in the liver and kidney lysosomal fraction, but in the kidney, the microsomal fraction was the main source of β-GL activity. The activity of β-GL was smallest in the blood plasma. The changes in the activity of β-GL were dependent on different kinds of antioxidant, organs and cellular fraction. These antioxidants were found to be highly responsible for the reactivity of β-GL in the liver and kidney. The least reactivity of the studied enzyme was shown in blood plasma. Injected antioxidants clearly induced the synthesis of the β-GL and increased the activity of β-GL dependent on the cellular fraction.