Evaluation of Mitochondrial Function and Antioxidant Enzyme Activities in CHO Cells Treated with Acetylsalicylic Acid

Gurgueira, S. A, Rossato, F. A. and Vercesi, A. E.

Departamento de Patologia Clínica, Faculdade de Ciências Medicas, UNICAMP, Campinas, SP, Brazil

Non-steroidal anti-inflammatory drugs (NSAID) have been proposed to act as pro-oxidants since above IC50 they are able to induce apoptosis in many types of cancer. NSAID can act as mitochondria uncouplers and they are also able to reduce neutrophils ROS production even though they do not act as ROS scavengers. Our purpose was to determine the effect of low doses of acetylsalicylic acid (ASA) on antioxidant enzyme activities and mitochondrial function. In this study, we evaluated the effect of ASA on: membrane potential (MP), oxygen consumption (OC), aconitase, citrate synthase, NADH dehydrogenase, MnSOD, glutathione peroxidase (GPx) and catalase (CAT) activities. These markers were tested in the presence or not of chronic H₂O₂-induced oxidative stress generated by the glucose oxidase during 24 h in CHO cells. Our preliminary results show that ASA (250 and 500 µM) induced oxidative stress in CHO cells since a significant decrease was detected on aconitase (p < 0.01) as well as on MnSOD (p < 0.01) and on CAT (p < 0.01) activities. When in the presence of 1 µM/h H₂O₂, aconitase activity remains decreased but MnSOD and CAT activities keep the same value as control. No changes were detected on MP, OC, citrate synthase, NADH dehydrogenase and GPx activities, and on cell viability (measured by LDH activity, Trypan Blue exclusion and annexin and 7-AAD). In conclusion, even low ASA concentrations are able to induce oxidative stress in CHO cells. H₂O₂, besides to keep MnSOD and CAT activities, did not protect these cells from ASA-oxidative stress.

Key words: acetylsalicylic acid, antioxidant enzymes, mitochondrial function
Supported by CNPq and FAPESP.