

## **Effect of Acetylsalicylic Acid on Antioxidant Enzyme Activities in HeLa Cells**

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Reactive oxygen species (ROS) generation in biological systems is a constant process and their levels can fluctuate even under normal conditions. Increase in ROS level can be prevented by antioxidant enzymes as superoxide dismutases (SOD), catalase (CAT) and glutathione peroxidases (GPx). Non-enzymatic antioxidants also contribute to ROS scavenging and minimization of oxidative stress. In this study, we evaluated the activity of the antioxidant enzymes MnSOD, GPx and CAT as well as aconitase activity as oxidative stress markers in experiments of chronic H<sub>2</sub>O<sub>2</sub>-induced oxidative stress in HeLa cells. Non-Steroidal Anti-inflammatory Drugs (NSAD) have been used as coadjuvant in the treatment of some cancer types. Our purpose was to determine the dose-response effect of acetylsalicylic acid (ASA) on antioxidant enzymes activities and on aconitase activity in the presence of chronic H<sub>2</sub>O<sub>2</sub>-induced oxidative stress. H<sub>2</sub>O<sub>2</sub> oxidative stress was induced by 10 μM/h H<sub>2</sub>O<sub>2</sub>, generated by the glucose oxidase/glucose system. This treatment significantly inhibited the activities of aconitase (p< 0.001), MnSOD (p<0.001), Se:GPx (p<0.001), and CAT (p<0.001). In the presence of ASA (100 μM to 3 mM), even in the absence of oxidative stress, there was a significant increase (p<0.001) on all antioxidant enzymes tested. MnSOD maximum activity was achieved with 300 μM ASA. For all the other enzymes the maximum activities was achieved with 2 mM ASA. In conclusion, ASA induces an increase in the activities of some antioxidant enzymes and it is a strong MnSOD stimulator. These results suggest it could be considered as a potential antioxidant since it can also protect the inhibition of antioxidant enzymes triggered by chronic H<sub>2</sub>O<sub>2</sub> generation.

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