POSTER 28

Study of oxidant and anti-oxidant molecular alterations in an eukaryotic model

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Mitochondrial dysfunction and protein aggregation are two phenomena that have been correlated with neurodegenerative diseases and are both interlinked with cellular oxidative stress. Mitochondrial dysfunction is associated with nucleic acid damage and protein aggregation with mistakes in protein folding. However, aggregation has not yet been defined as a cause or consequence of neurodegenerative diseases. Therefore, this thesis' main objectives were to analyze the influence of different concentrations of hydrogen peroxide, an oxygen-free radical produced by our cells, in protein aggregation and genetic material as well as the role of vitamin E as an antioxidant agent. For that purpose, S. cerevisiae cellular samples were collected throughout the exponential growth phase. Analysis of insoluble aggregates was performed using the electrophoretic technique SDS-PAGE followed by protein lysis and DNA breakage bands through 1,5% agarose electrophoresis followed by RAPD-PCR. Exposure to the oxidizing agent over time has enhanced protein aggregation, namely ovalbumin. However, the anti-oxidant did not appear to reverse the oxidizing effect and seems to have a pro-oxidant effect. Although differences in protein aggregation were observed over time, there were no breaks in the genetic material.

Keywords: Neurodegenerative diseases, DNA damage, protein aggregation, oxidative stress.