Redox mechanisms are involved in several processes, such as cell proliferation, survival and differentiation, among other ways by modulating kinases, phosphatases and transcription factors activity that can occur through reversible oxidative modification of cysteine residues. We are interested in redox processes underlying osteogenic differentiation induced by BMP-2, using MC3T3-E1 cell lineage. Our objective is to investigate redox modifications as possible modulators of the osteogenic differentiation process. We performed qPCR analysis to quantify gene expression, western blotting and redox western blotting assays for protein quantitation, phosphorylation and redox status, and Alizarin Red assays to verify calcified extracellular matrix deposition. We characterized osteogenic differentiation in MC3T3-E1 cells, upon BMP2 treatment, through gene expression of osteogenic markers Runx2, Osteocalcin and Osterix, SMAD1 (belonging to the BMP-2 pathway) protein phosphorylation, and calcified extracellular matrix deposition over time. Our data showed that BMP2 treatment resulted in NOX4 up regulation, which probably also lead to hydrogen peroxide production. Besides, we have investigated peroxiredoxin modulation in this process, and no significant change in peroxiredoxin 1 and 2 expression levels was detected, but peroxiredoxins could be found oxidized after BMP2 treatment, in a dose response manner. In vitro analysis showed that cells exposed to N-acetyl-L-cysteine and PEG-catalase, display impaired osteogenic differentiation, detected by lower levels of calcified extracellular matrix deposition compared to non-treated cells. Furthermore, N-acetyl-L-cysteine and PEG-catalase treatments did not affect cell survival and proliferation. Moreover, phosphorylation of SMAD 1 protein, as well as overall protein tyrosine phosphorylation levels are reduced under these redox treatments. Our data suggest that redox pathways can modulate cell signaling during osteogenic differentiation process.

**Key Words:** BMP2, osteogenic differentiation, redox signaling

**Acknowledgements:** CEPID/Redoxome; CEPID/Human Genome and Stem Cell Research Center; FAPESP; CNPq