

Type II metacaspase mediates light-dependent programmed cell death in *Chlamydomonas reinhardtii*

Among the crucial processes that preside over the destiny of cells from any type of organism, are those involving their self-destruction. If this process is very well characterized and conceptually logical to understand in multicellular organisms like animals and plants, the levels of knowledge and comprehension of its existence are still quite enigmatic in unicellular organisms. In our lab, we use *Chlamydomonas reinhardtii* to lay the foundation for understanding the mechanisms of programmed cell death (PCD), in a unicellular photosynthetic organism. In a nitrosative stress context induced by S-nitrosoglutathione (GSNO), we recently showed that while PCD induces the death of a proportion of cells, it allows the survival of the remaining population. A quantitative proteomic analysis aiming at unveiling the proteome of PCD in *Chlamydomonas*, allowed us to identify key proteins deregulated during PCD that led to the discovery of essential mechanisms. We show that in *Chlamydomonas*, PCD relies on the light dependence of a photosynthetic organism to generate reactive oxygen species (ROS), here singlet oxygen, and induce cell death. Finally, we have obtained and characterized mutants for the two metacaspase genes present in *Chlamydomonas*, and showed for the first time that a type II metacaspase is essential for PCD execution.

Keywords: *Chlamydomonas reinhardtii*, metacaspase, programmed cell death, light, nitric oxide, singlet oxygen, nitrosative stress