Reactive oxygen species (ROS) and dynamic intracellular reductive/oxidative (redox) balance are critical for normal cellular functions. Disruption of redox homeostasis leads to aberrant cell signaling, macromolecules damage and is associated with human pathologies such as neuro-degenerative diseases and cancers.

Our team has been conducting research to better understand redox regulation, oxidative stress response mechanisms, and ROS induced genome instability and cell death. We use two complementary experimental systems, human cell lines and the yeast *S. cerevisiae* to investigate these mechanisms.
Our current research activities focus on the following projects. The project I is to study the biological function of oxidative stress response system (Figure 1), including peroxiredoxins, thioredoxins and glutathione regarding genome stability maintenance and cell death regulation in yeast and in human cells. These elements are not only the first line of defence against deleterious effect of oxidative stress but also actively involved in redox signaling. More recently, we focused our effort on characterizing the subcellular compartments redox environments and their dynamic regulations in various biological processes (Figure 2). The project II is to understand the physiological role of the S. cerevisiae protein complex Tah18/Dre2 and to define its biochemical
properties. This protein complex exhibits functional interaction with the DNA polymerase delta and may be implicated in linking intracellular redox states to cell fate. We wish to understand more precisely how this complex is linked to DNA replication via iron-sulfur (Fe-S) biosynthesis.

The project III aims to exploit redox-modulating strategies as a therapeutic approach. Therapeutic selectivity in cancer therapy could be achieved by ROS-mediated mechanisms based on the different redox states in normal and malignant cells.

We are studying whether and how a rational redox modulation could have selective and synergistic effects together with treatment by drugs or ionizing radiations in cancer cells.
Figure 2: Targeting a yellow fluorescent protein-based redox sensor (rYFP) to the cytosol, nucleus and mitochondrial matrix of HeLa cells (upper panel) and time course analysis of compartment-specific redox changes in response to H2O2 treatments (lower panel).