

What yeast can tell us about how cells commit suicide?

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Multicellular organisms developed a complex system to balance cell proliferation and cell death in order to guarantee correct embryonic development and tissue homeostasis. Failure of cells to undergo programmed cell death (PCD) can potentially lead to severe diseases, including neural degeneration, autoimmunity and cancer. Identifying the molecules involved in PCD and understanding the regulation of the process are crucial for prevention and management of these diseases. Evidence of the enormous impact of PCD, of which apoptosis is the most frequent morphological phenotype, on human health makes it one of the today's main research topics. Since PCD was initially considered specific of metazoans, biological models were first restricted to animal cells. Actually, based on the absence of known crucial PCD regulators, as indicated by plain homologies searches, as well as on the difficulty to explain the sense of cell suicide in a unicellular organism, it was not accepted that these organisms could possess a PCD mechanism. However, evidence has been reported in the last decade indicating that the process of self-destruction in different unicellular organisms, namely in yeast, can also take place.

In the present communication, I will present the research we have been developing on PCD, based on the exploration/exploitation of yeast as a simple eukaryotic unicellular model system. Particular focus will be given to our more recent studies suggesting a complex regulation and interplay between mitochondria and the vacuole in acetic acid induced PCD. The validation in mammalian cell lines of the hypothesis postulated with the yeast model will be also discussed.