PREFACE

Both autophagy and programmed cell death (PCD) are fundamental processes of cellular maintenance that are closely interrelated in plant and animal cells under physiological and stressful conditions. Differentiation, immune response, lack of nutrients, and wide range of abiotic factors induce their development and realization of survival or cell death scenario. Microtubular cytoskeleton is known as one of the principle players in the mediation of PCD/autophagic signals. Chapter One in this book presents an overview of the current knowledge about the role of MTs in PCD- and autophagy-related processes in plants. Chapter Two reviews the mechanisms and consequences of virus interactions with the hotel cell-death machinery, to help understand potentially pathologically relevant consequences that will help in the design of intervention strategies and the development of antiviral therapies. The final chapter discusses the control of the levels of different reactive oxygen species (ROS) and their interaction with hormones and transcriptional factors in relation with programmed cell death in leaves.

Chapter 1 – Both autophagy and programmed cell death (PCD) are fundamental processes of cellular maintenance that are closely interrelated in plant and animal cells under physiological and stressful conditions. Differentiation, immune response, lack of nutrients, and wide range of abiotic factors induce their development and realization of survival or cell death scenario. Microtubular cytoskeleton is known as one of the principle players in the mediation of PCD/autophagic signals. Microtubules (MTs) were shown involved in numeral PCD-like processes as well as in autophagosome maturation and traffic. As very dynamic structures MTs are highly sensitive to external stimuli and their functional state is basic for their role in realization of pro-death or pro-recycling cellular program. Generally, it provided with

cytoskeletal network reorganization via balancing between stable/dynamic MTs behavior that is closely depends on variety of post-translational modifications of tubulin and its situational protein microenvironments. This chapter represents an attempt to overview the current knowledge about the role of MTs in PCD- and autophagy-related processes in plants.

Chapter 2 – In order to thwart a viral infection, the host has developed an integrated defense network that includes innate and adaptive immunity as well as regulated cell death (RCD), which appears to represent an ancestral defence mechanism for the host. Indeed, RCD is important to control several aspects of the cell fate in various situations, from embryonic development and adult tissue homoeostasis development, to stress response, including viral infection, where different but complementary highly regulated arms of the complex RCD process exist and can be activated, with various consequences. In order for a host to cope with a viral infection, RCD can play a critical role in the elimination of virus-infected cells and/or to control the cell-to-cell viral dissemination to protect the whole organism. During the pathogen-host co-evolution, viruses have developed various strategies to counteract defenses of the host and, among other things, they have acquired the capacity to subvert host-cell RCD. For some viruses, disabling host-cell RCD, at least for some time, could represent an important step in the replication cycle as an effort to prolong their own survival and possibly establish a persistent infection, which can lead to chronic diseases. On the other hand, other viruses may take advantage of RCD to facilitate shedding and disseminate more efficiently in the host or to prevent the presentation of viral antigens to the immune cells. By encoding numerous proteins that either are homologs to cellular apoptosis-regulatory factors or interfere with cellular proteins that control RCD-related pathways, several different viruses are able to induce or modulate the different cellular pathways that control RCD. Virus modulation of cell death participates in the pathological process associated with several human diseases; therefore understanding mechanisms and consequences of virus interactions with the host cell-death machinery is essential to understand potentially pathologically relevant consequences that will help in the design of intervention strategies and the development of antiviral therapies.

Chapter 3 – The leaf is a complex organ composed of different tissues and cells that is subjected to a variety of environmental factors affecting the course of its aging, senescence and death. Most investigations on leaf death have been focused on the senescence of photosynthetic mesophyll cells, whose characteristic loss of chlorophyll becomes the reference symptom preceding leaf death. Autumnal and monocarpic leaf deaths are developmental process

genetically programmed but, at the cell level, many changes that precede them are similar to those of the senescence of shaded basal leaves and even to those of the stress-dependent leaf aging. Most knowledge of the molecular processes in programmed leaf death has been gained through the investigation with the last two deaths, provoked by developmental and environmental factors that in strict sense are not programmed leaf death. However, analogies with processes in programmed cell death in animals provide unifying views where reactive oxygen species (ROS, mainly singlet oxygen $^1O_2$, anion radical superoxide $O_2^{\bullet -}$ and hydrogen peroxide H_2O_2) emerge as key intracellular signals. The steady state level of each ROS depends on generation and scavenging activities closely linked, respectively, to the photosynthetic electron transport and to systems that protect cell from the destructive action of ROS and, frequently, the destructive actions of ROS are hardly distinguishable from their signaling role in cell death. By controlling the level of different ROS, chloroplasts regulate leaf death and assume similar roles to several ones attributed to mitochondria in programmed animal cells. High ratios ($^1O_2$ plus $O_2^{\bullet -}$)/H_2O_2 seem key to shot the autocatalytic cell path to death where hormones and transcriptional factors joint to change the feed-back regulation of cell processes to a feed-forward spiral of deleterious reactions. In this chapter, the authors will discuss the control of the levels of different ROS and their interaction with hormones and transcriptional factors in relation with programmed cell death in leaves.