EDITORIAL FOREWORD

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'Apoptosis Review Series'

Apoptosis: a mapped path to cell death

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Guest Editor

One of the most fascinating, dynamic and fast evolving disciplines in biology is that of cell death. Cell death research encompasses not only the study of programmed forms of cell death (both apoptosis and autophagic cell death), necrosis and other modes of cellular demise, but also the role of these phenomena in physi-ological and pathological processes including development, aging and disease. This field now boasts to over 200,000 publications, new approaches and methodologies as well as a new vocabulary, making it a rich and diverse research area.

The cell death field has attracted much attention, mainly because of its relevance to development, degenerative diseases and cancer. During the past 20 years apoptosis has received extraordinary attention. However, the field of cell death research is by no means a new phenomenon [1]. The concept of cell death and the terminology has been evolving since the 19th century. The mid-1900s witnessed a surge in interest in the field and the application of systematic approaches to study this phenomenon. The term *programmed cell death* refers to controlled or regulated forms of death associated with a series of biochemical and morphological changes [2–4]. The realization that some forms of cell death were biologically controlled or programmed, led to exploitation of these processes and has made profound impact in various fields of medicine [5–7].

Nowadays, programmed cell death is synonymous with apoptosis, however, based on the original definition it also refers to autophagic cell death [8]. The term *Apoptosis* was first used to describe a particular morphology of cell death [9], common to the vast majority of physiological cell deaths. This morphology includes shrinkage and blebbing of cells, rounding and fragmentation of nuclei with condensation and margination of chromatin, shrinkage and phagocytosis of cell fragments without accompanying inflammatory responses [9–11]. The morphology of cells undergoing apoptosis appeared dissimilar and distinct from the morphology associated with necrosis [9, 10]. *Necrosis*, a term commonly used by pathologists, refers to any deaths associated with the loss of control of ionic balance, uptake of water, swelling and cellular lysis [12, 13]. This lysis releases many intracellular constituents, attracting immune cells and provoking an inflammatory response.

In 1980, apoptosis became the focus of attention, primarily because of the relative ease at which it could

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be distinguished morphologically from other types of cell death. Within a few years apoptosis and delineation of the underlying biochemical and molecular pathways dominated cell death research. The discovery of the Bcl-2 family of proteins [14] death receptors [15, 16], caspases [17, 18] and a role for mitochondria [19, 20] in apoptosis were just a few major milestones in the history of the field. Today the morphological and biochemical changes associated with apoptosis is largely explained by activation of caspases, and apoptosis has become generally accepted as caspase-dependent programmed cell death [21].

The Journal of Cellular and Molecular Medicine has shown a long-standing interest in the subject and is committed to advancement in apoptosis research. The current review series is intended to review the milestones in the history of the field and implications for health and disease.

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