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Editorial

A Programmed Cell Death: Apoptosis

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1. Editorial

Apoptosis is a programmed process of cell death. It is used to eliminate unwanted cells in the early stages of development. For example between the fingers of a developing hand in adults, apoptosis is used to remove irreparably damaged cells from the body. Apoptosis also plays a role in the prevention of cancer. Prevention of apoptosis for any reason can lead to uncontrolled cell division and subsequent tumours development. Programmed cell death, especially apoptotic cell death, occurs under physiological conditions and is also desirable under pathological conditions. However, the more we learn about the cell signaling cascade, the less likely we are to find a constrained, well confined signaling pathway. In this context, a comprehensive description of signaling pathway connections is needed to identify the most important regulatory molecules and select the most appropriate therapeutic target. On the other hand, irregularities in programmed cell death signaling pathways often lead to tumours development, and cancer-related mortality is expected to continue to increase despite efforts to develop more active and selective antitumor compounds increase. Programmed Cell Death (PCD; sometimes called cellular suicide) is cell death as a result of intracellular events such as apoptosis and autophagy. PCD is usually performed in a biological process that benefits the life cycle of an organism.

Indeed, tumours cell plasticity poses a major challenge in chemotherapy, and improved cancer treatment appears to depend on the appropriate combination of drugs. Cell death usually occurs in a regulated manner under physiological and pathological conditions. Apoptosis, historically called necrobiosis or chromatolysis and long used as a synonym for programmed cell death, is an ever expanding field. The more we know about crosstalk and organelle regulation, the less effective simple classifications are. Pure and watertight cell death pathways no longer make sense. Classically, surface cell death receptors and mitochondria have been considered important sites for the induction of apoptosis. Nonetheless, recent studies have revealed novel regulated pathways that emphasize the importance of other organelles in the regulation of apoptosis. Cell death is usually regulated under physiological and pathological conditions. Apoptosis, historically called necrobiosis or chromatolysis and long used as a synonym for programmed cell death, is an ever expanding field. The more you know about crosstalk and organelle regulation, the less effective the simple classification will be. The pure and watertight cell death pathway is no longer meaningful. Classically, surface cell death receptors and mitochondria have been considered important sites for the induction of apoptosis. Nonetheless, recent studies have revealed new regulated pathways that emphasize the importance of other organelles in the regulation of apoptosis.

Alzheimer's disease is a human specific disease. Even our closest primate relatives do not develop a medical condition, not to mention the clinical outcomes that can be considered real AD. Nevertheless, much of our understanding of the evolution of amyloid pathology has progressed through studies in various animal models. There are no animal models that accurately reflect every aspect of AD, but there are many models of Aβ deposition. In general, these can be divided into models in which amyloid pathology evolves spontaneously with age and genetically modified mice that express variants of the APP. Animals with naturally occurring amyloid deposits are attractive in that researchers do not suffer from the many

warnings associated with genetically engineered mice, such as: b. Isolate the contribution of overexpression to the model phenotype and the introduced mutation. However, animals that experience amyloid deposits as a result of normal aging have a significantly longer lifespan than rodents (very long for some non-human primates) and are costly, so their use can be difficult to justify.