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Apoptosis: Editorial on Programmed Cell Death

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Editorial

In multicellular organisms, apoptosis is the process of programmed cell death (PCD). Characteristic cell changes (morphology) and death are the result of biochemical events. Blabbing, cell shrinkage, nuclear fragmentation, chromatin condensation, and chromosomal DNA fragmentation are examples of these alterations. It is now assumed that cells are driven to affirmatively commit suicide in a developmental setting, whereas in a homeostatic context, the absence of specific survival cues may give the impetus for suicide [1]. The shape and biochemistry of these suicide pathways appear to vary, with some following the road of "apoptosis" and others following a more generalised path to deletion, but both being genetically and synthetically motivated. Although there is evidence that certain signs of "apoptosis," such as endonuclease activation, can be generated without triggering a genetic cascade, real apoptosis and programmed cell death must likely be genetically regulated. It's also becoming obvious that mitosis and apoptosis are related in some way, and that the balance attained is determined by signals received from suitable growth or survival factors [2].

Autophagy is a cytoplasmic process characterised by the creation of giant vacuoles that consume organelles in a precise order before the nucleus is destroyed. Macro autophagy, also known as autophagy, is a catabolic process in which bulk cytoplasmic contents, aberrant protein aggregates, and excess or damaged organelles are degraded through autophagosomic-lysosomal degradation. Autophagy is triggered by a lack of nutrients, but it has also been linked to physiological and pathological processes like development, differentiation, neurodegenerative disorders, stress, infection, and cancer [3].

Apoptosis initiation is closely regulated by activation pathways, because once apoptosis has started, the cell will surely die. The intrinsic pathway (also known as the mitochondrial pathway) and the extrinsic pathway are the two most well-known activation mechanisms. Intracellular signals generated when cells are stressed activate the intrinsic pathway, which is dependent on the release of proteins from mitochondria's inter membrane gap [4]. Extracellular ligands connect to cell-surface death receptors, triggering the extrinsic pathway, which results in the creation of the death-inducing signalling complex (DISC).

In reaction to a stimulus, a cell initiates intracellular apoptotic signalling, which may result in cell suicide. Nuclear receptor binding by glucocorticoids, heat, radiation, nutrient deprivation, viral infection, hypoxia, increased intracellular concentration of free fatty acids, and increased intracellular calcium concentration, for example, caused by membrane damage, can all cause a damaged cell to release intracellular apoptotic signals. A variety of biological components, including poly ADP ribose polymerase, may also play a role in apoptosis regulation. Single cell variations have been seen in stress-induced apoptosis experiments.

Apoptotic signals must cause regulatory proteins to activate the apoptosis pathway before enzymes can initiate the actual process of cell death. If the cell no longer needs to die, this phase allows those signals to cause cell death or to terminate the process. Several proteins are involved, but there are two major ways to regulate them: targeting mitochondrial functioning or directly transducing the signal to apoptotic processes via adaptor proteins. An increase in calcium concentration within a cell produced by pharmacological activity has been found as an extrinsic mechanism for initiation in various toxin investigations, which can also trigger apoptosis via the calcium binding protease calpain.

Loss of regulation of cell death (excess apoptosis) might, on the other hand, result in neurological illnesses, hematologic diseases, and tissue damage. In neurodegenerative disorders such as Alzheimer's and Parkinson's, neurons that rely on mitochondrial respiration incur apoptosis (a discovery known as the "Inverse Warburg hypothesis"). Furthermore, neurological disorders and cancer have an inverse epidemiological comorbidity. Excess, uncontrolled apoptosis is intimately associated to HIV development. The amount of CD4+ lymphocytes in a healthy person is balanced with the cells generated by the bone marrow; however, in HIV-positive patients, this equilibrium is disrupted due to the bone marrow's inability to renew CD4+ cells. When CD4+ cells are activated, they die at a faster rate due to uncontrolled apoptosis [5].

Defects in signalling networks that regulate the Bcl-2 family proteins can cause hyperactive apoptosis at the molecular level. Increased expression of apoptotic proteins like BIM, or decreased proteolysis of these proteins, causes cell death and can result in a variety of diseases, depending on the cells where BIM is overactive. Cancer cells can avoid apoptosis by using mechanisms that inhibit BIM expression or by increasing BIM proteolysis.

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