



Various Histological Patterns for Causing Cell Damage or Cell Death

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Description

Cell theory, cellular illness, and the ensuing advancements and applications are covered in this chapter. Studies on cell biology, cell pathology, and cell damage are linked together, which is highlighted. The significance of technical developments in histology, biochemistry, and molecular approaches for cell biology and cell pathology research is also acknowledged. Special attention is placed on the work of Rudolph Virchow and his former students in the creation of the cell theory in biology and pathology and produced a complete characterization of apoptosis, so giving momentum to the modern area of cell damage research. Cell injury research is still a significant and productive area of continuing study and development.

Two histological patterns of cell death in animals with graft-versus-host disease brought on by intravenous injection of allogeneic bone marrow were seen in the colonic intestinal epithelium. Animals and spleen cells were taken 2 to 6 hours after receiving 9 Gy of whole-body radiation before being sacrificed 25 days later. Colonic interstitial cells with oncosis-like ultrastructure may be seen in the electron micrographs from a mouse graft-versus-host disease model. According to reduction in cytoplasmic density and blebbing of the basement membrane, cells exhibit nuclear contraction with chromatin condensation and cytoplasmic edoema (pyknosis).

The various forms of cell damage and death

Apoptosis: Apoptosis, often referred to as shrinkage necrosis, is a type of planned cell death that is brought on by the activation of the caspase enzyme. A genetically designed series of molecular changes occurs in tandem with distinctive morphological changes, such

as the shrinking of dying cells, and results in phagocytic absorption of the dying cell before the rupture of the plasma membrane.

Oncosis/Necrotic cell death: Oncosis, also known as necrotic cell death, is a kind of cell death characterised by swelling of the organelles and cells as a result of a loss of integrity in the cell membrane, and the release of components into the extracellular space, which results in an inflammatory reaction. During acute cell damage, necrosis seems to develop randomly. Recent research has however demonstrated that necrosis may also be brought on deliberately and in response to particular stimuli.

Necroptosis: Necroptosis is a kind of programmed cell death characterised by organellar and cell enlargement as well as a loss of cell membrane integrity. It is brought on by certain stimuli and occurs through a specific signalling route (programmed necrotic cell death).

Pyroptosis: Inflammasomes, which are macromolecular complexes that are put together in response to various irritants, activate caspase 1 and caspase 11 (which are different from the caspases that mediate apoptosis) to cause pyroptosis, a kind of cell death. Pyroptosis results in cell membrane rupture, much like necrotic cell death. Interleukin-1 and interleukin-18, two important pro-inflammatory cytokines produced by the caspase-1-mediated processing of their cytoplasmic precursors, are also linked to it.

Death of autophagic cells: Dysregulated or excessive autolysis, a collection of intracellular processes that result in the breakdown of cellular components, causes autophagic cell death. It has a role in both clinical states and developmental processes.