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Unlocking the Potential of Apoptotic Cells in Disease and Therapy

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Introduction

Apoptosis, the programmed and highly regulated process of cell death, is an essential mechanism for maintaining cellular balance and organismal health. Through apoptosis, the body eliminates damaged, aging, or unneeded cells without triggering inflammation, ensuring the integrity of surrounding tissues. This process is critical during embryonic development, immune system regulation, and tissue homeostasis. A hallmark of apoptosis is the formation of apoptotic bodies, which are membrane-enclosed vesicles containing cellular remnants and bioactive molecules that play a pivotal role in cellular communication and immune modulation. The significance of apoptosis extends beyond normal physiology into the realm of disease and therapy. Dysregulated apoptosis is a key feature of numerous pathological conditions, including cancer, autoimmune diseases, and neurodegenerative disorders. In cancer, for instance, defective apoptotic pathways enable malignant cells to evade death and grow uncontrollably, while excessive apoptosis in neurodegenerative diseases leads to the loss of functional neurons. Moreover, apoptotic bodies are emerging as potential biomarkers and mediators of intercellular communication, influencing immune responses and tissue repair mechanisms. Advances in biomedical research have shed light on the therapeutic potential of targeting apoptotic pathways and harnessing apoptotic bodies for disease management. From developing drugs that restore apoptosis in cancer cells to using apoptotic bodies as vehicles for drug delivery, the therapeutic opportunities are vast. This article explores the dual role of apoptotic cells in disease and therapy, highlighting their potential as diagnostic tools, therapeutic targets, and innovative treatment modalities.

Description

Bone marrow mesenchymal immature microorganisms are non-hematopoietic undifferentiated organisms with the limit with regards to self-restoration and multipotent separation that keep up with bone marrow homeostasis. MSCs can separate into osteoblasts, adipocytes, fibroblasts, chondrocytes and non-mesenchymal cell types. To regulate immune responses, MSCs can also inhibit the proliferation and function of several major immune cells, including dendritic cells, T and B lymphocytes and natural killer cells. As a result, MSCs have been identified as a potential source of cells for immune therapies and tissue regeneration. MSCs maintain their stem cell properties by reusing multiple cellular factors from apoptotic bodies and phagocytosing apoptotic bodies, as demonstrated in this study [1].

A typical exosome is surrounded by a phospholipid membrane that contains detergent-resistant membrane domains (lipid rafts) and lipids typical of their cellular origin, such as cholesterol, sphingomyelin and ceramide. Lipid raft-associated proteins like flotillin and glycosylphosphatidylinositol-anchored proteins are also present. Exosomes have higher concentrations of certain lipids than their parent cells, enhancing the rigidity of the exosomal membrane. Exosome-specific proteins, such as Alix and tumor susceptibility gene 101 (TSG101), are components of the ESCRT complex that play a role in MVB biogenesis. The presence of tetraspanins, such as CD9, CD63, CD81 and CD82, is yet another distinguishing feature of exosomes. Exosomes also contain cytosolic proteins like Rabs, which help to promote exosome docking and membrane fusion, as well as annexins, which are thought to control the dynamics of the membrane cytoskeleton and membrane fusion [2]. Numerous studies have demonstrated that exosomes contain nucleic acid cargo that is functionally active when released into recipient cells. Non-coding RNAs, such as microRNA and long non-coding RNA (lncRNA), tRNA fragments, small-interfering RNAs, structural RNAs, small RNA transcripts and RNA-protein complexes, may be included in this nucleic acid cargo. Exosomes are excellent biomarkers because, in addition to various RNA species, they contain DNA that could represent the entire genome and genomic mutations. There have been reports of mitochondrial DNA as well as chromosomal DNA [3].

Microvesicles The direct outward budding of the plasma membrane of living cells results in the release of membrane microvilli, which in turn produces microvesicles (MVs) or microparticles (MPs) ranging in size from 50 to 1000 nm. These vesicles typically have a diameter of up to 1000 nm, but smaller vesicles with a diameter of 50 nm also emerge from the plasma membrane. Various shapes of microvesicles have also been reported. Integrins, selectins and CD40 are typical MV-detection markers. However, depending on the type of cell from which they are secreted, various other markers may be used. Additionally, studies suggest that microvesicles are the vesicles that sediment at less than 10,000–20,000 x g. The lipid composition of microvesicles is uniformly distributed across the bilayer membrane, in contrast to the asymmetrical distribution that is found on the two leaflets of the plasma membrane, since microvesicles are shed by the budding of the plasma membrane. Even though cells shed MVs when they are at rest, some cells release MVs depending on the stimulant they receive. Phorbol esters, calcium, purinergic receptors and P2Y receptors are all thought to be involved in the robust release of MVs [4].

At Human Metabolome Technologies America (HMT) and Metabolon Inc. (Durham, NC), sample extraction, processing, compound identification, curation and metabolomic analyses were performed. Before being shipped for metabolomic analysis, sequential centrifugation was used to briefly separate supernatants from cell pellets. For HMT;After being spiked with internal standards in 10 ul of water, the supernatant samples were filtered through a 5-kDa cut-off filter to get rid of small vesicles and macromolecules. CE-TOFMS was used to measure cationic compounds using positive ion mode ESI. CE-MS/MS was used to measure anionic compounds in either the positive or negative ion mode of ESI. The CE-QqQMS analysis was improved by diluting the samples. Migration time, mass to charge ratio and the peak area normalized to the internal standard and standard curves were used to identify and quantify metabolites. The reported concentrations were calculated backwards based on the number of cells used in the experimental setup and they are expressed as a percentage of a million cells [5].

Recovery standards were added to the samples for untargeted metabolomics analysis using Metabolon to keep an eye on the quality control of the analysis. Methanol precipitated the samples over two minutes with shaking. After that, the organic solvent was removed from the samples by

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placing them on the TurboVap and the samples were kept O/N in nitrogen. The samples were examined under four distinct conditions: two for positive ion mode ESI analysis by two distinct reverse phase (RP)/UPLC-MS/MS methods, one for negative ion mode ESI analysis by RP/UPLC-MS/MS and one for negative ion mode ESI analysis by HILIC/UPLC-MS/MS.

Conclusion

The study of apoptotic cells and their bodies has unveiled a vast and dynamic frontier in disease biology and therapeutic innovation. These cellular remnants, once dismissed as byproducts of programmed cell death, are now understood as powerful mediators of cellular communication and immune regulation. Their role in maintaining physiological balance underscores their importance in health, while their dysregulation reveals their critical involvement in a wide array of diseases, from cancer and autoimmune conditions to neurodegenerative disorders. Targeting apoptotic pathways offers a promising strategy for developing innovative therapies. In cancer, reactivating apoptosis can halt tumor progression, while in neurodegenerative diseases, modulating apoptotic mechanisms may preserve neuronal integrity. Apoptotic bodies, with their ability to encapsulate and transport bioactive molecules, represent a novel avenue for drug delivery, biomarker discovery, and therapeutic monitoring.

As research continues to deepen our understanding of apoptosis, the potential to translate these insights into clinical applications grows exponentially. By harnessing the dual nature of apoptotic cells—as both contributors to disease and instruments of therapy—medicine stands poised to address some of the most pressing health challenges of our time. The ongoing exploration of this field promises a future where the mechanisms of cell death become a cornerstone for life-saving treatments, bridging the gap between fundamental biology and transformative healthcare solutions.

Acknowledgment

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Conflict of Interest

None.

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