Mini Review
Volume 12:7, 2022

Journal of Bioengineering & Biomedical Science

ISSN: 2155-9538 Open Access

The Apoptotic Cell and its Body: Potentials for Disease Message and Therapeutic Target

Fazhi Qi*

Department of Plastic Surgery, Zhongshan Hospital, Fudan University, Shanghai 200032, PR China

Abstract

Apoptosis is commonly referred to as programmed cell death that does not cause inflammation. Numerous studies have focused on the intricate nature of apoptosis, accumulating a wealth of information that has led to effective disease treatments as well as a deeper comprehension of the fundamental process. The traditional apoptosis regulatory factors and intrinsic and extrinsic signaling pathways have been clearly defined. Since ancient times, apoptosis-based drugs and treatments have been used to treat disease. In clinical settings, small-molecule apoptosis inducers have been used to treat diseases like cancer by killing off abnormal cells. Recombinant proteins and antibodies, which have improved apoptotic efficacy and selectivity, are the subject of extensive research and the FDA has granted approval to some of them. Apoptotic bodies (ApoBDs) are membrane-bound vesicles that are derived from the disassembly of apoptotic cells. Prior to being discovered to be capable of delivering useful materials to healthy recipient cells (such as autoantigens), these tiny sealed sacs containing information and substances from dying cells were regarded as garbage bags. With a focus on apoptosis-related therapeutic applications and ApoBDs, this review summarizes and discusses current knowledge of apoptosis.

Keywords: Apoptosis • Apoptotic bodies • Caspase • Death receptors • Drug design • Intercellular communication

Introduction

It is necessary for over 50 billion cells to undergo apoptosis each day in the human body to maintain tissue homeostasis. Apoptosis is programmed cell death that involves distinct cell shrinkage, chromatin condensation and plasma blebbing1. Numerous neurological disorders, including Alzheimer's, Parkinson's and Huntington's, are exacerbated by excessive apoptosis. On the other hand, autoimmune diseases, such as systemic lupus erythematosus (SLE), also known as lupus, in which the body's immune system mistakenly attacks healthy tissue in many parts of the body, may be linked to absence of apoptosis. Numerous apoptotic bodies containing a variety of cellular components, including proteins, lipids, DNA, microRNAs and mRNAs, are produced during apoptosis. Macrophages, dendritic cells, epithelial cells, endothelial cells and fibroblasts consume apoptotic bodies, which are then internalized, consumed and degraded in the lysosomes. It is hypothesized that apoptotic bodies may facilitate intercellular communication through the transfer of cellular factors because engulfment of apoptotic cells may prime macrophages to generate molecular memory. However, it is unknown whether apoptotic processes and body production regulate stem cell function.

Description

Bone marrow mesenchymal immature microorganisms (MSCs) 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

***Address for Correspondence:** Fazhi Qi, Department of Plastic Surgery, Zhongshan Hospital, Fudan University, Shanghai 200032, PR China; E-mail: dr.qifazhi@126.com*

Date of Submission: 01 July, 2022, Manuscript No. jbbs-22-80886; **Editor Assigned:** 03 July, 2022, PreQC No. P-80886; **Reviewed:** 11 July, 2022, QC No. Q-80886; **Revised:** 16 July, 2022, Manuscript No. R-80886; **Published:** 23 July, 2022, DOI: 10. 37421/2155-9538.2022.12.311

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 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

Metabolite identification could be carried out with reverse scores between the experimental data and authenticated standards by employing a library that is based on authenticated standards and contains the retention time/index (RI), mass to charge ratio (m/z) and chromatographic data on all of the molecules in the library (including MS/MS spectral data). This library is called Metabolon. Biochemicals can be identified by utilizing all three data points, even though there may be similarities based on one of these factors. On request, the R code used to create heatmaps and volcano plots can be obtained.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

References

1. Fuchs, Yaron and Hermann Steller. "Programmed cell death in animal development and disease." *Cell* 147 (2011): 742-758.
2. Rothlin, Carla V., Eugenio A. Carrera-Silva, Lidia Bosurgi and Sourav Ghosh, et al. "TAM receptor signaling in immune homeostasis." *Annu Rev Immunol* 33 (2015): 355.
3. Lindsten, Tullia, Andrea J. Ross, Ayala King and Wei-Xing Zong, et al. "The combined functions of proapoptotic Bcl-2 family members bak and bax are essential for normal development of multiple tissues." *Mol Cell* 6 (2000): 1389-1399.
4. Gregory, Christopher D. and John D. Pound. "Cell death in the neighbourhood: Direct microenvironmental effects of apoptosis in normal and neoplastic tissues." *J Clin Pathol* 223 (2011): 178-195.
5. Ryoo, Hyung Don, Travis Gorenc and Hermann Steller. "Apoptotic cells can induce compensatory cell proliferation through the JNK and the wingless signaling pathways." *Develop Cell* 7 (2004): 491-501.

How to cite this article: Qi, Fazhi. "The Apoptotic Cell and its Body: Potentials for Disease Message and Therapeutic Target." J Bioengineer & Biomedical Sci 12 (2022): 311.