Journal of Biochemistry and Cell Biology

Perspective Open Access

A Brief View on Mitotic Catastrophe

Zhivotovsky*

Division of Toxicology, Institute of Environmental Medicine, Sweden

Mitotic catastrophe (MC) has long been considered as a mode of cell death that results from premature or inappropriate entry of cells into mitosis and can be caused by chemical or physical stresses. Whereas it initially was depicted as the main form of cell death induced by ionizing radiation, it is today known to be triggered also by treatment with agents influencing the stability of microtubule, various anticancer drugs and mitotic failure caused by defective cell cycle checkpoints. Although various descriptions explaining MC exist, there is still no general accepted definition of this phenomenon. Here, we present evidences indicating that death-associated MC is not a separate mode of cell death, rather a process ('prestige') preceding cell death, which can occur through necrosis or apoptosis. The final outcome of MC depends on the molecular profile of the cell.

During mitosis, proliferating cells undergo several structural and molecular changes, characterized by chromatin condensation, spindle formation, nuclear envelope fragmentation and cytoskeleton reorganization. Chromosome segregation is carried out by complex machinery – the mitotic spindle that is based on a bipolar array of microtubules [1]. Microtubules are highly dynamic polymers that continuously grow and shrink, and in the spindle, this behaviour is regulated by proteins that bind to the sides or ends of microtubules. The rate of microtubule disassembly, from a state of polymerization to depolymerisation, is designated as the catastrophe rate.

Mitotic catastrophe is characterized by an unscheduled or prolonged activation of mitotic drivers followed by cell death. In some cases, permanent cell cycle arrest following the aberrant mitosis is also regarded as an outcome of mitotic catastrophe [2]. Causes of mitotic catastrophe include unscheduled entry into mitosis from interphase or prolonged mitotic arrest caused by the activation of the spindle-assembly checkpoint.

A clear definition of the cell death mechanism termed "mitotic catastrophe" has been continuously under development over the past decade and beyond. As a broad definition, it is currently accepted that mitotic catastrophe is cell death occurring during mitosis ensuing from DNA damage or other aberrations affecting the mitotic process, such as malfunctions in the mitotic machinery [3]. This pathway to cell death is quite unique in both mechanism and molecular constituents, although commonalities and overlap with other cell death mechanisms, specifically apoptosis and necrosis, do exist. Accurate detection of mitotic catastrophe in the laboratory is essential to distinguish it from other modes of cell death. Creative techniques used to this end are constantly being explored [4]. Mitotic catastrophe is a critical process in the prevention of genomic instability and possible ensuing malignancies. An overview of the cell cycle and the role of mitotic catastrophe as an integral part of this process.

Mitotic catastrophe has also been described as a delayed form of reproductive death based on observations that the multinucleated giant cells can be temporarily viable. The term 'reproductive death' denotes the loss of the ability of a cell to generate viable progeny that reproduced continuously. As most of the cells undergoing MC eventually die, cellular processes that lead to irreversible growth arrest and those that are termed 'reproductive death' may better fit to conditions known as senescence. Senescent cells are generally characterized by a reduction in proliferative capacity, adoption of a flattened and enlarged cell shape and an increase of β-galactosidase (SA-β-gal) activity. Although senescence was associated with MC, it has been shown that the polyploidy giant cells expressing senescent marker (SA-β-gal activity) may overcome the state of growth arrest and even undergo de-polyploidization.

Mitotic catastrophe (MC) has long been considered as a mode of cell death that results from premature or inappropriate entry of cells into mitosis and can be caused by chemical or physical stresses. Whereas it initially was depicted as the main form of cell death induced by ionizing radiation, it is today known to be triggered also by treatment with agents influencing the stability of microtubule, various anticancer drugs and mitotic failure caused by defective cell cycle checkpoints [5]. Although various descriptions explaining MC exist, there is still no general accepted definition of this phenomenon. Here, we present evidences indicating that death-associated MC is not a separate mode of cell death, rather a process ('prestige') preceding cell death, which can occur through necrosis or apoptosis. The final outcome of MC depends on the molecular profile of the cell.

Cells that fail to execute an apoptotic program in response to mitotic failure are likely to divide asymmetrically in the next round of cell division, with the consequent generation of aneuploidy cells. This implies that disabling of the apoptotic program may actually favour chromosomal instability, through the suppression of mitotic catastrophe. Mitotic catastrophe thus may be conceived as a molecular device that prevents aneuploidization, which may participate in oncogenes, is. Mitotic catastrophe is controlled by numerous molecular players, in particular, cell-cycle-specific kinases (such as the cyclin B1-dependent kinase Cdk1, polo-like kinases and Aurora kinases), cell-cycle checkpoint proteins, survivin, p53, capsizes and members of the Bcl-2 family.

References

1. Vakifahmetoglu H, Olsson M, Zhivotovsky B (2008) Death through a Tragedy: Mitotic Catastrophe. Nature 15: 1153-1162.
2. Ianzini F, Mackey MA (1997) "Spontaneous Premature Chromosome Condensation and Mitotic Catastrophe Following Irradiation of Hela S3 Cells". Int JRadiatBiol 72: 409-421.
3. Castedo M, Perfettini JL, Roumier T, Andreau K, Medema R, et al. (2004) "Cell Death by Mitotic Catastrophe: A Molecular Definition". Oncogene 23: 2825-2837.

***Corresponding author:** Zhivotovsky, Division of Toxicology, Institute of Environmental Medicine, Sweden, E-mail: zhivoto765@gmail.com

Received: 03-Mar-2022, Manuscript No: JBCB-22-146, **Editor assigned:** 05-Mar-2022, Pre QC No: JBCB-22-146 (PQ), **Reviewed:** 19-Mar-2022, QC No: JBCB-22-146, **Revised:** 24-Mar-2022, Manuscript No: JBCB-22-146 (R), 31-Mar-2022, DOI: 10.4172/jbcb.1000146

Citation: Zhivotovsky (2022) A Brief View on Mitotic Catastrophe. J Biochem Cell Biol, 5: 146.

4. Korsnes MS, Korsnes R (2017)"Mitotic Catastrophe in BC3H1 Cells Following Yessotoxin Exposure". Front Cell Dev Biol 5: 30.

5. Erenpreisa J, Kalejs M, Ianzini F, Kosmacek EA, Mackey MA, et al. (2005)"Segregation of Genomes in Polyploid Tumour Cells Following Mitotic Catastrophe". Cell Biol Int 29: 1005-1011.