



## Role of Murine Embryonic Stem Cells in Apoptosis

Jason Perry\*

Department of Paediatric Surgery, Tulane University Health Sciences Center, Louisiana, USA

### DESCRIPTION

The inner cell mass of blastocysts gives rise to pluripotent murine Embryonic Stem (mES) cells. All the tissues of the embryo proper are produced by a very small number of ES cells. Due to the limited population size, ES cells need to have highly effective mechanisms to protect DNA from harm, to repair it when it does, and to stop the spread of any mutations that result from it. One effective method to stop the spread of mutations is apoptosis, and after UV-induced DNA damage, mES cells are known to be significantly more adept at apoptosis than differentiated cells. In mES cells, ionising radiation appears to be a less powerful apoptotic trigger [1]. The effectiveness of basic stress defences, specifically antioxidant defences in mES cells, is still poorly understood. Because they are a constant result of regular metabolism, Reactive Oxygen Species (ROS) are a primary cause of DNA damage. Due to the accelerated production of ROS from the mitochondrial respiration chain as the ambient oxygen pressure rises, the majority of differentiated cells are sensitive to it. In normal human fibroblasts, for instance, levels of intracellular peroxides and protein carbonyls rose and lipofuscin developed more quickly when the ambient oxygen partial pressure was raised from 21% to 40% [2].

Simultaneously, the replicative lifespan shrank to a relatively small number of Population Doublings (PDs), while the rate of telomere shortening multiplied by a factor of 4 to 10. Murine somatic cells that have undergone differentiation are even more vulnerable to oxidative stress than human cells. Because of this, murine fibroblasts age prematurely under typical *in vitro* circumstances even though their telomeres are longer than those of human cells and they produce telomerase, protecting them. These cells can proliferate without clear boundaries if oxidative damage is reduced much like telomerase-expressing human fibroblast under usual circumstances. However, genetic stability is not the same as immortality [3]. In fact, oxidative stress and time both increase the frequency of mutations and genomic instability in differentiated mouse cells. They hypothesised that in order to maintain high levels of genomic stability at the single-

cell level, mES cells should be more capable of withstanding stress than differentiated ones.

They think that learning more about how ES cells are able to keep their genomes stable over numerous cell divisions will help they understand how somatic stem cells age *in vivo* and how that affects age-related diseases in the adult body [4]. On gelatin-coated culture flasks in conditions containing leukaemia Inhibitory Factor (LIF), mES cells can be cultivated in culture seemingly without limit. Growth in medium lacking LIF enables differentiation into cell types that no longer possess the ability to develop indefinitely, form Embryoid Bodies (EBs), and eventually senescence. Using this method, they and others have shown that one of the crucial events occurring early in ES cell differentiation is the down regulation of telomerase reverse Transcriptase (Tert), the catalytic subunit of telomerase. The ability of mES cells to repair strand breakage caused by oxidative stress and their tolerance to this damage are presently being examined. Antioxidant defence skills are quite strong in mES cells; however they become less strong during the early stages of differentiation into EBs. In 3T3 fibroblasts and mouse embryonic fibroblasts, ROS levels are still significant. Additionally, mES cells repair DNA strand breaks more effectively than differentiated mouse 3T3 fibroblasts. They have discovered numerous potential antioxidant and stress-resistance genes that are downregulated during ES development using microarrays and validation by Reverse Transcription-Polymerase Chain Reaction (RT-PCR) [5].

### REFERENCE

1. Malhas A, Lee CF, Sanders R, Saunders NJ, Vaux DJ. Defects in lamin B1 expression or processing affect interphase chromosome position and gene expression. *J Cell Biol.*2022; 176: 593-603
2. Mattout A, Pike BL, Towbin BD, Bank EM, Gonzalez-Sandoval A, Stadler MB, et. al. An EDMD mutation in *C. elegans* lamin blocks muscle-specific gene relocation and compromises muscle integrity. *Curr Biol.*2023; 21: 1603-1614

**Correspondence to:** Jason Perry, Department of Paediatric Surgery, Tulane University Health Sciences Center, Louisiana, USA, Email: Jason.p@email.com

**Received:** 30-Dec-2022, Manuscript no: JSCRT-22-19806, **Editorial assigned:** 02-Jan-2022, Pre QC no: JSCRT-22-19806(PQ), **Reviewed:** 18-Jan-2022, QC no: JSCRT-22-19806, **Revised:** 23-Jan-2022, Manuscript no: JSCRT-22-19806(R), **Published:** 30-Jan-2022, DOI: 10.35248/2157-7633.23.13.576.

**Citation:** Perry J (2023) Role of Murine Embryonic Stem Cells in Apoptosis. *J Stem Cell Res Ther.* 13:576.

**Copyright:** © 2023 Perry J. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

3. Meshorer E, Yellajoshula D, George E, Scambler PJ, Brown DT, Misteli T. Hyperdynamic plasticity of chromatin proteins in pluripotent embryonic stem cells. *Dev Cell*. 2010; 10: 105-116
4. Melcer S, Hezroni H, Rand E, Nissim-Rafinia M, Meshorer E. Histone modifications and lamin A regulate chromatin protein dynamics in early embryonic stem cell differentiation. *Nat Commun*. 2019; 3: 910-911
5. Bhattacharya D, Talwar S, Mazumder A. Spatio-temporal plasticity in chromatin organization in mouse cell differentiation and during *Drosophila* embryogenesis. *Biophys J*. 2019; 96: 3832-3839