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Editorial

Molecular Signalling Pathways Regulating Cell Survival, Death and Differentiation

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Cellular development and cell fate specification are precise events regulated by different mechanisms that cooperate to maintain the correct pattern of gene expression. Accordingly, extracellular signals including changes in the microenvironment and mechanical stimuli are able to trigger the fine regulation of these transcriptional networks to control cell fate decisions such as survival, proliferation, or differentiation.

This Special Issue includes original articles concerning the biochemical and molecular biology of cell survival, death, and differentiation, particularly those focusing on regulation of the transcriptional network by the extracellular microenvironment.

In particular, Huang and collaborators [1] explored the effects of phosphodiesterase-5 (PDE-5) silencing in bone marrow mesenchymal stem cells (MSCs) on high glucose (HG)-induced myocardial fibrosis and cardiomyocyte apoptosis. The authors demonstrated that silencing PDE-5 in MSCs can decrease HG-induced myocardial fibrosis and cardiomyocyte apoptosis by activating the cGMP/PKG pathway and may play a role in preventing and treating diabetic cardiomyopathy.

MSCs were also the focus of study in the paper by Innamorati *et al*. [2], in which the role of intracellular cAMP in promoting neural differentiation in chorion-derived cells was investigated. Morphological and protein expression analyses revealed that cAMP elevation activated the cAMP response element-binding protein (CREB) thereby triggering a preliminary step towards neuronal differentiation of chorion-derived MSC. However, like other MSCs, the stimulus was insufficient to promote stable differentiation.

In contrast, Helvoort Lengert and collaborators [3] studied testicular germ cell tumors (TGCTs), the most frequent tumours of teenagers and young men, focusing in particular on cisplatin (CDDP)-resistant forms. NanoString technology revealed several differentially expressed genes related to DNA repair and cell cycle regulation on CDDP-resistant cell lines compared to parental cell lines. Moreover, the proteasome inhibitor MG-132 demonstrated cytotoxic activity in all cell lines evaluated, even enhancing cell lines' sensitivity to CDDP. Overall, the data suggest that a targeted therapy based on proteasome inhibition may contribute to overcome acquired chemotherapy CDDP-resistance.

Finally, Yuan *et al*. [4] investigated the mechanisms involved in the regulation of Japanese encephalitis virus (JEV) by the long non-coding RNA (lncRNA)-SUSAJ1. In this paper, the authors demonstrated that lncRNA-SUSAJ1 promoted the expression of pro-apoptotic genes and triggered cellular oxidative stress. Moreover, lncRNA-SUSAJ1 promoted apoptosis via the endoplasmic reticulum stress response, thereby inhibiting viral replication. The findings of this study provide insight into the antiviral effect of lncRNA-SUSAJ1 on JEV.

In summary, this Special Issue collects relevant papers investigating intracellular pathways related to mesenchymal stem cell survival and differentiation, as well as molecular mechanisms related to apoptosis in testicular cancer cells and in encephalitis virus.

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Conflict of Interest

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