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Stem cells differentiation and probing their therapeutic applications in hematological disorders

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Numerous lines of evidence support that bone marrow is a rich source of stem cells that can be used for research purposes and to treat some complex blood diseases and cancers. Stem cells are mother cells that posses the capacity to become any type of cell in the body. Stem cells are cells without specific structure and characterized by their ability to self-renew or multiply while maintaining the potential to develop into other types of cells. Stem cells can normally become cells of the blood, heart, bones, skin, muscles or brain. Although, there are different sources of stem cells, all types of stem cells have the same capacity to develop into multiple types of cells. Stem cells are generally described as unspecialized cells with unlimited proliferation capacity that can divide (through mitosis) to produce more stem cells. Several types of adult stem cells have been characterized and can be cultured in vitro, including neural stem cells, hematopoietic stem cells, mesenchymal stem cells, cardiac stem cells and epithelial stem cells. They are valuable as research tools and might, in the future, be used to treat a wide range of diseases such as hematological hereditary diseases, Parkinson's disease, diabetes, heart disease and many other diseases. Currently, two types of stem cells have been identified based on their origin, namely embryonic stem cells and adult stem cells. Collectively, although many literatures have studied stem cell application in terms of clinical practice, therapeutic stem cell still at infancy stage.

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AKT2 and CCAAT/enhancer-binding protein β transcription factor (C/EBP- β) mediate SphK1 up-regulation in imanitib-resistant K562 cells

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In our previous study, we have shown that SphK1 is up-regulated in imatinib-resistant CML cells by a pathway contingent on a phosphoinositide 3-kinase/AKT2/mammalian target of rapamycin signaling pathway. In order to better understand this mechanism, we examined the signaling pathways responsible for transcriptional up-regulation of SphK1in imatinib-resistant CML cells. Pharmacological and molecular approaches demonstrated that only activation of AKT2, in addition to the CCAAT/enhancer-binding protein-β transcription factor is involved in transcriptional up-regulation of SphK1 in imanitib-resistance CML cells. Our data implicate AKT2 as an important mediator signaling in imanitib resistance leading to up-regulation of SphK1 and point to SphK1 and sphingosine-1-phosphate production as potential therapeutic targets in CML

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