OMICS International
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Hulya Cabadak, J Blood Disord Transfus 2015, 6:5
http://dx.doi.org/10.4172/2155-9864.C1.011

3rd International Conference on

Hematology & Blood Disorders

November 02-04, 2015 Atlanta, USA

Non-neuronal cholinergic system in megacaryocytic differentiated K562 cells

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Acetylcholine is a neurotransmitter in the nervous system but it serves also as a paracrine or autocrine factor in different cell types, where it is linked to functions like proliferation and cell differentiation. Muscarinic receptors are relatively abundant in the central nervous system and peripheral parasympathetic nervous system. Many cells express a mixture of muscarinic receptor transcripts. Changes in muscarinic M2 and M3 receptor mRNA levels in response to agonist treatment have been reported in cerebellar granule cells, Chinese hamster ovary cells, lymphocytes and in the human neuroblasto macellline SH-SY5Y. Several researchers have suggested that non-neuronalacetylcholine and cholinergic agonists alter cell growth and proliferation of lymphocytes. Costa et al. demonstrated that acetylcholine released from T-lymphocytes acts via the M3 acetylcholine muscarinic receptor (mAChR) to trigger nuclear signaling and up-regulation of gene expression in T and B-lymphocytes. We have previously demonstrated the presence of M2, M3 and M4 mAChRs and M3 subtype mediated NO signaling in K562 chronic myelogenous leukemic cells. We also showed that carbacho (CCh), cholinergic agonist, treatment leads to changes in muscarinic M2, M3 and M4 receptor transcripts as well as M2 and M3 protein levels and enhances cyclic adenosine monophosphate (cAMP) accumulation in K562 cells. In this study, we investigated the levels of muscarinic receptor protein expression in megacaryocytic differentiated K562 leukemia cells. And we also investigated the effects of agonist stimulation on megacaryocytic differentiated K562 leukemia cells proliferation. Muscarinic cholinergic receptors activate stimulatory growth mechanisms in megacaryocytic differentiated K562 leukemia cells. stimulation on cell proliferation and expression in megacaryocytic differentiated K562 leukemia cells. We also found that CCh changed proliferation of megacaryocytic differentiated K562 cells proliferation in 24 hour, these results suggest that CCh modulates megacaryocytic differentiated K562 leukemic cells proliferation through muscarinic acetylcholine receptors. We showed that CCh-treatment leads to changes in muscarinic M2, M3 and M4 receptor transcripts as well as M2 and M3 protein levels.

Biography

Hulya Cabadak is an Associate Professor at Marmara University, Turkey. Her research areas are Cancer Research, Cell Biology and Biology.

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