OMICS International
conferenceseries.com

J Cancer Sci Ther 2014, 7:10
http://dx.doi.org/10.4172/1948-5956.C1.061

8th Euro Global Summit on Cancer Therapy

November 03-05, 2015 Valencia, Spain

Cross talk of the first-line defense TLRs with PI3K/Akt pathway, in preconditioning therapeutic approach

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Toll-like receptor family (TLRs), pattern recognition receptors, is expressed not only on immune cells but also on non-immune cells, including cardio-myocytes, fibroblasts and vascular endothelial cells. One main function of TLRs in the non-immune system is to regulate apoptosis. TLRs are the central mediators in hepatic, pulmonary, brain and renal ischemic/reperfusion (I/R) injury. Up-regulation of TLRs and their ligation by either exogenous or endogenous danger signals play critical roles in ischemia/reperfusion–induced tissue damage. Conventional TLR-NF-κB pathways are markedly activated in failing and ischemic myocardium. Recent studies have identified a cross talk between TLR activation and the PI3K/Akt pathway. The activation of TLRs is proposed to be the most potent preconditioning method after ischemia, to improve the cell survival via the mechanism involved the PI3K/Akt signaling pathway and to attenuate the subsequent TLR-NF-κB pathway stimulation. Thus, TLRs could be a great target in the new treatment approaches for myocardial I/R injury.

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The suppressive effect of organism senescence on cancers

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Most scientific literature reports that aging favors the development of cancers. Each type of cancer, however, initiates and evolves differently and their natural history can start much earlier in life before their clinical manifestations. The incidence of cancers is spread throughout human life span, and is the result of pre- and post-natal aggressions, individual susceptibility, developmental changes that evolve continuously throughout an individual's life, and time of exposure to carcinogens. Finally, during human senescence, the incidence declines for all cancers. Frequently, the progression of cancers is also slower in aged individuals. There are several possible explanations for this decline at the tissue, cell and molecular levels, which will be described. It is time to ask why some tumors are characteristic of either the young, the aged, or during the time of a decline in the reproductive period and finally, why the incidence of cancers declines late during senescence of human beings. These questions need to be addressed before the origin of cancers can be understood.

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