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Retinol/vitamin a signaling, stem cells and cancer stem cells

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Natural and synthetic derivatives of retinol, the alcohol form of vitamin A are generally associated with cell differentiation via its metabolite retinoic acid. Contrary to this, we have demonstrated a novel function of retinol in the self-renewal of embryonic stem (ES) cells by activating PI3 kinase signaling pathway via IGFII/IGF1 receptor axis in retinoic acid independent mechanism. Our studies have shown that ES cells do not express the critical retinol metabolizing enzymes and receptor proteins such as STRA6 and CRBP indicating a complete depletion of retinol metabolism in stem cell a property that may be shared by all stem cells including cancer stem cells (CSCs). Retinol supports self renewal of undifferentiated ES and induced pluripotent stem (iPS) cells in the absence of mouse embryonic feeder cells that offers a powerful tool to generate clinically relevant patient specific pluripotent cells for regenerative medicine. An impairment of retinol metabolizing machinery has been reported in many breast cancers. In a significant advancement, a homogenous self renewing population of putative cancer stem cells (CSCs) was created from a mouse mammary tumor. The cells exhibit typical characteristics of stem cells such as unlimited growth, expression alkaline phosphatase and Nanog. These cells are important to investigate the signaling mechanisms of unregulated growth and resistance to therapies and to define genetic profile of CSC to identify novel biomarkers for targeted CSCs without killing the normal breast stem cells for long-term cure of cancer.

Biography

Dr. Khillan is Associate Professor at the University of Pittsburgh at Pennsylvania. His research is focused on the mechanisms of self renewal of stem cells and the mechanisms of tumorigenesis by breast cancer stem cells. He has published more than 50 papers in reputed journals. He is also Director of Transgenic and Gene Targeting Facility.