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The stem cell origin of cancer

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In this presentation, evidence is provided that in each major theory of the origin of cancer: field theory, chemical carcinogenesis, infection, mutation or epigenetic change, the tissue stem cell is involved in the generation of cancer. The stem cell theory of cancer proposes two major concepts: 1) that cancers arise from stem cells that are present in the tissues of mature adult individuals, and 2) that cancers are composed of the same types of cells as normal tissues, i.e., stem cells, transit amplifying cells and terminally differentiated cells. The hypothesis that cancers arise due to maturation arrest of stem cell differentiation was proposed by us in 1994 for all tissues, based primarily on observations of the origin of teratocarcinomas and hepatocellular carcinomas. About the same time, tumor transplantation studies led to renewed interest in the concept that cancers were maintained by cells that had the properties of stem cells. In this presentation, the author will document the involvement of stem cells as the cells of origin of cancer in each of the major theories of the origin of cancer using selected example of cancers and from this provide models that depict the nature of the cells in a cancer. Although the cancer cells may be identified as more differentiated cells in the cancer cell lineage or hierarchy (transit amplifying cells), the property of malignancy or the molecular lesion of the cancer exists in the cancer stem cell. In the case of teratocarcinomas, normal germinal stem cells have the potential to become cancers if placed in an environment which allows expression of the cancer phenotype (field theory). In the cases of cancer due to chemically induced mutations, virus infections, mutations or epigenetic changes, the molecular lesion or infection occurs in the tissue stem cells. Cancer stem cells give rise to transit amplifying cells and terminally differentiated cells similar to what happens in normal tissue renewal. However, the major difference between cancer growth and normal tissue renewal is that the cancer transit amplifying cells fail to differentiate normally and accumulate (maturation arrest), whereas normal transit amplifying cells systematically differentiate and die.

Biography

Stewart Sell is a graduate of the College of William and Mary and the University of Pittsburgh Medical School. He did a residency in Pathology at the Massachusetts General Hospital and worked at NIH and the University of Birmingham, England in Immunology. He has served on the faculty of the University of Pittsburgh, University of California at San Diego, U. Texas, Houston, and Albany Medical College. He has published over 400 papers and 12 books in immunology, pathology, infectious diseases, cancer biology and stem cells. He was the first to show that a population of lymphocytes had surface immunoglobulins, that lymphocytes could be activated to proliferate by reaction with this surface Ig and that the activation had to pass through a critical G1 block. By examining cellular changes and alphafetoprotein production during different models of chemical hepatocarcinogenesis, he was the first to conclude that there were tissue stem cells in the adult liver and that these stem cells could give rise to liver cancer. He is presently determining how different risk factors interact to increase the risk for liver cancer and the contribution of stem cells to cancer, as well as the nature of the immune response to influenza and asthma in mouse experimental models.

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