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Cancer stem-cells in oral cancer and pre-cancer

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The traditional stochastic clonal expansion model for cancer hypothesized that a single cell having acquired mutations for transformation into a cancer cell yielded bulk of the primary tumor which comprised of cells having heterogenic potential. "Cancer stem cell hypothesis" postulates that cancer heterogeneity is theresult of mutations that renders a normal stem cell cancerous, or cause a cancerous cell to develop stem cell-like characteristics. Cancer stem cells (CSC), like the somatic progenitor counterpart are capable of self-renew, differentiation into heterogeneous population, have modified anti-apoptotic properties, and are resistant to chemotherapy and radiation. In 2003, Clarke et al. first identified CSC in solid tumors, namely breast cancer. In 2007, Prince et al. identified subpopulation of head and neck carcinoma (HNSCC) cells that had CSC-like phenotype. Liu et al. in 2011 proposed the horizontal hierarchal model of CSC consisting of precancerous CSC, primary CSC, migrating CSC, and chemo-radio-resistant CSC. Traditional cancer therapies do not target CSC, and being chemo-radio-resistant they could be playing a major role in post-surgical recurrence and metastasis. Researchers are unsure of the utility of CSC surface and intra-cellular markers they studied, as their expression varied with the type of cancer and as different researchers obtained different results with the same bio-marker. Understanding CSC shall enhance our knowledge with regards to cancer initiation, progression, response to therapy, metastasis, and recurrence.

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