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STEM CELL VERSUS THE DE-DE-DIFFERENTIATION HYPOTHESES OF HUMAN CARCINOGENESIS: ADULT HUMAN ORGAN-SPECIFIC STEM CELLS AS TARGETS FOR CANCER STEM CELLS

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Human carcinogenesis appears to fit the initiation, promotion, progression concept that involves multiple steps and mechanisms. The two major, but opposing, hypotheses of the origin of this process, by which one normal cell is initiated to start this complex evolutionary process, include the stem cell hypothesis and the de-differentiation or re-programming hypothesis. With the isolation of human stem cells (embryonic and organ specific adult stem cells), as well as the induced pluripotent stem cells (iPS), arguments have been made to support both competing hypotheses. The basic assumption to be made in this presentation is that there appears to be more direct evidence to support the hypothesis. Since every human organ contains organ-specific adult stem cells, a single rare normal adult stem cell can be converted from a cell capable of either symmetrical or asymmetrical cell division to one that can not divide asymmetrically, by an error-prone DNA repair process or by an error prone DNA replication process, to become initiated. This single initiated adult organ-specific stem cell, if exposed to epigenetic agents, such as pollutants, drugs, cytokines, hormones, growth factors, at threshold levels, for regular and sustained fashion, in the absence of antioxidants, can be promoted to become independent of these promoters and to be transformed to an invasive and metastatic cancer stem cells. Evidence will be presented from the isolation and characterization of a human breast adult stem cell, having expressed *Oct4A, ABCG-2* genes, but having no expressed *Connexin* genes or having no function gap junctions. These cells will be shown to give rise to human breast cancer stem cells. Moreover, these normal or cancer stem cells will be shown to form 3D organ-specific organoids that can be used to screen for both tumor promoters and chemo-preventive agents.

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