

[Fifth AACR International Conference on Frontiers in Cancer Prevention Research, Nov 12-15, 2006]

Invited Abstract: Targeting Stem/Progenitor Phenotypes for Cancer Prevention

Bone Marrow-Derived Stem Cells: Implications for Cancer Prevention.

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Abstract

PL03-02

Cancer chemoprevention has focused for many years on directly inhibiting the growth of epithelial cells that are believed to give rise to cancer. Carcinogenesis is a multi-step process, and recent work strongly suggests that the target cells for transformation are pluripotent stem cells or early progenitors. The cancer stem cell theory has important implications with respect to both chemotherapy approaches as well as for chemoprevention, since efforts at inhibiting the promotion of initiated stem cells in selected individuals could in theory reduce cancer rates. The epithelial model of cancer has raised for consideration the epithelial stem cell as a primary candidate for the precursor of cancer stem cells. Stem cells have generally been thought to possess unique properties of longevity, self-renewal and pluripotentiality. However, we now recognize that many aspects of stem cell behavior are governed not by the stem cell itself but by surrounding stromal cells, known as the stem cell niche. In addition, several studies now suggest that in some model systems, solid tumors may arise not from epithelial stem cells but from bone marrow-derived stem cells (BMDCs) that are recruited to the site by chronic inflammation and tissue injury. The discovery that BMDCs can give rise to epithelial tumors was made using a *Helicobacter*-dependent mouse model of gastric cancer [Houghton J 2004]. Work from several laboratories had shown that chronic inflammation leads to mobilization of BMDCs, which can then be recruited and engrafted to the sites of chronic injury. When recruited to the stomach, for example, BMDCs take on the appearance of epithelial cells, losing hematopoietic markers and in their place expressing epithelial genes. In this context, the BMDCs serve as a second line of defense to aid the healing process, but in the setting of a "wound that will not heal", the BMDC may end up replacing the tissue stem cell and can then progress to malignancy. The recognition that circulating BMDCs can engraft in epithelial tissues, take on many of the characteristics of epithelial cells and then give rise to solid tumors, offers a new and different perspective on the process of carcinogenesis. In the BMDC model, cancer initiation is not due simply to a genetic mutation but to the initial process of engraftment of BMDCs in the damaged epithelium.

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Chemopreventive agents could be targeted to reducing BMDC mobilization and engraftment, and toward restoring the proper stem cell niche and preventing conversion to cancer stem cells.

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