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The role of mesenchymal stem cells in tumor growth and spread

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Currently, there are many promising clinical trials using mesenchymal stem cells (MSCs) in cell-based therapies of diseases ranging widely from graft-versus-host to joint and cartilage disorders. Increasingly, however, there is a concern over the clinical use of MSCs because they are also known to track to tumors, and once resident in the tumor microenvironment (TME) to support tumor growth and spread. For instance, we established that MSCs in the ovarian TME promoted tumor growth and favored angiogenesis. We also recently reported that distinct *ex vivo* priming of MSCs polarizes them into an anti-tumor *MSC1* phenotype or a pro-tumor *MSC2* phenotype. Here we aimed to determine how the polarized MSC phenotypes affect tumor growth and spread. Our *central hypothesis* is that *MSC1* will track to TMEs and shift the balance from a tumor promoting stroma to a tumor eradicating one that attenuates tumor growth and spread by influencing the secretion of defined soluble factors and extracellular matrix proteins. Indeed, tumor spheroids and colony forming assay results supported that notion. *MSC1* delivered to mice with established ovarian tumors attenuated tumor growth and spread when compared to animals treated with MSCs, *MSC2,* or vehicle controls**.** The discrete chemokines, cytokines, and extracellular matrix components present in tumor stroma following the interaction of the polarized MSCs with tumors were also measured. Migration and invasion assays confirmed the influence of the tumor-resident polarized cells on the tumor's metastatic potentials. This information should considerably help in the design of safer MSC-based therapies.

Biography

Dr. Betancourt obtained her Ph.D. from Georgetown University in 1992. Her research training began as a post-doctoral fellow with Dr. Stuart Yuspa (NIH, NCI) a pioneer in the development of murine models of multi-step carcinogenesis. This experience led to a post-doctoral position in the Tulane Cancer Center to study the role of mesenchymal stem cells (MSCs), low oxygen (hypoxia) and environmental stress factors (alarmins) in cancer. Her current research efforts have led to several significant observations: "Alarmins" secreted from tumors recruit bone marrow-derived mesenchymal stem cells MSCs; Toll-like receptors (TLRs) on MSCs drive this recruitment; Elevated secretion of the alarmin, LL-37 from ovarian tumors recruits MSCs to support the growth and spread of the tumor; and She recently published paradigm-shifting evidence that MSCs undergo polarization into both the accepted anti-inflammatory MSC2 phenotype and a newly described pro-inflammatory MSC1 phenotype. Based on this newly described MSC paradigm into both MSC1 pro-inflammatory and MSC2 anti-inflammatory phenotype, she has filed an invention disclosure (US 61/391,749) and is in the process of filing various patent applications to protect the methodology involved and the potential for targeted stem cell-based therapies using these phenotypes in partnership with Wibi+Works, LLC. She has published more than 25 peer-reviewed journal articles and serves as an editorial board member for the Journal of Stem Cell and Therapy and the World Journal of Stem Cells.