The role of tumor-associated macrophages (TAMS) and micro RNA 146 alpha in cancer survival and efferocytosis in the tumor microenvironment

In this study, the role of the tumor microenvironment on cancer cell survival was analyzed. The ability of cancer cells to inhibit nitric oxide production in macrophages and the increased drug resistance capabilities of the cancer cells in the presence of macrophages in the tumor microenvironment was explored. These recruited macrophages, also known as tumor-associated macrophages, appear to give the cancer cells drug resistance which can lead to failure in chemotherapy. The hypotheses were tested using Griess reagent systems to measure nitric oxide and trypan blue cell viability assays. The results showed a decrease in cancer cell death in the presence of macrophages when exposed to an anti-cancer drug cisplatin. Nitric oxide levels produced by cancer cells appeared lower when cultured with macrophages. MicroRNA-146a suppresses prostate cancer transformation from androgen-dependent to -independent cells, suppresses a kinase coding gene which reduces cell proliferation, invasion, and metastasis to human bone marrow endothelial cell monolayers, and is dysregulated by latent membrane protein 1 (LMP1) which contributes substantially to the oncogenic potential of Epstein-Barr virus. It is projected that microRNA-146a and other microRNAs may one day become biomarkers for clinical diagnosis and prognosis of several types of cancer including prostate. The miR-146a expression profile was investigated using novel African American and Caucasian prostate cell lines representing each pathological stage: benign, androgen dependent and independent tumors. Relative miRNA expression was determined by qRT-PCR, miRNA plate assay and smart flare technology after isolating total RNA from the cells and the exosomes from the tumor microenvironment. Our initial findings shows that this micro RNA is upregulated in several cancer cell lines there by reducing NO production in them and the neighbouring Macrophages(by exosome delivery) and there by giving these cancer cells survival advantage.

ACKNOWLEDGEMENT: This research is supported by a grant from the Borroughs Wellcome Fund, NSF-LSAMP, NIH-MARC and a Disability Supplement from NIH-NCI.

Biography
Hirendra Nath Banerjee recieved his BS with honors and MD degree from Calcutta University, India. A MS in Molecular Biology from Conolly College of Pharmacy and Health Sciences at Long Island University, NY and Ph.D. in Molecular Biology from Howard University Cancer Center, Washington, D.C. Dr. Banerjee did his post doctoral training at Yale University Medical School and Medical University of South Carolina before joining Elizabeth City campus of the University of North Carolina where he is now a tenured Professor in the department of Natural Pharmacy and Health Sciences. Dr. Banerjee did a sabbatical at Rockefeller University under the mentorship of nobel laureate Dr. Gunter Blobel in studying the cell biology of nuclear pore complex’s. Dr. Banerjee's current research involves cancer biology and Efferocytosis.

Notes: