

## Early and Late Damages in Chromosome 3 of human lymphocytes after radiation exposure

Mayumi Sunagawa<sup>1,2</sup>, Lingegowda Mangala<sup>1,3</sup>, Ye Zhang<sup>1,4</sup>, Munira Kahdim<sup>5</sup>, Bobby Wilson<sup>2</sup>, Francis A. Cucinotta<sup>1</sup> and Honglu Wu<sup>1</sup>

<sup>1</sup>NASA Johnson Space Center, Houston, TX

<sup>2</sup>Texas Southern University, Houston, TX

<sup>3</sup>University of Houston Clear Lake, Houston, TX

<sup>4</sup>Wyle Laboratories, Houston, TX

<sup>5</sup>Oxford Brooks University, Oxford, UK

Tumor formation in humans or animals is a multi-step process. An early stage of cancer development is believed to be genomic instability (GI) which accelerates the mutation rate in the descendants of the cells surviving radiation exposure. GI is defined as elevated or persistent genetic damages occurring many generations after the cells are exposed. While early studies have demonstrated radiation-induced GI in several cell types as detected in endpoints such as mutation, apoptosis and damages in chromosomes, the dependence of GI on the quality of radiation remains uncertain. To investigate GI in human lymphocytes induced by both low- and high-LET radiation, we initially exposed white blood cells collected from healthy subjects to gamma rays *in vitro*, and cultured the cells for multiple generations. Chromosome aberrations were analyzed in cells collected at first mitosis post irradiation and at several intervals during the culture period. Among a number of biological endpoints planned for the project, the multi-color banding fluorescent in situ hybridization (mBAND) allows identification of inversions that were expected to be stable. We present here early and late chromosome aberrations detected with mBAND in chromosome 3 after gamma exposure. Comparison of chromosome damages in between human lymphocytes and human epithelial cells is also discussed.