

Seminar Announcement

Novel Mechanisms of Genetic Instability in Cancer



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DNA can adopt alternative structures that do not conform to the Watson-Crick B DNA helix (i.e. non-B DNA structures), including hairpins, cruciforms, H-DNA, and Z-DNA. Sequences that can adopt non-B DNA structures are very abundant in human genomes, and importantly, non-B DNA-forming sequences often co-localize with mutation hotspots in the human genome. For example, non-B DNA-forming sequences co-localize with translocation breakage hotspots in human cancer genomes, implicating non-B DNA in cancer etiology. Dr. Vasquez's lab, and others, have shown that non-B DNA structures can cause genomic instability, and can alter DNA metabolism (e.g. DNA transcription, replication, and repair). However, the mechanisms involved in these processes are not well characterized. Non-B structures cause distortions in the double helix, and therefore, may be recognized as a form of a DNA "damage" by repair proteins and initiate a DNA damage response involving multiple repair mechanisms. For example, Dr. Vasquez's lab has shown that nucleotide excision repair (NER) and mismatch repair (MMR) proteins are involved in non-B DNA-induced mutagenesis in yeast and mammalian cells. Results of such studies will be discussed.

Friday, November 4th, 2016

11:00 a.m. to 12:00 p.m.

Venue: Room GL-100

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