CANDIDATE STATEMENT FOR
ASSOCIATE DIRECTOR OF CONSTITUENCY

Name: Dr. Marijo Kent-First

Present Position: Assistant Professor
Department of Biological Sciences
Organization: Florida A&M University

Chapter Affiliate: National Affiliate
(Currently resurrecting Florida A&M University Chapter)

Professional Biography: Currently Dr. Kent-First serves as Assistant Professor in the Department of Biological Sciences at Florida A&M University where she teaches a range of classes and engages undergraduate and graduate students in research aimed at determining the mechanism of genomic instability in ROS induced cell lines in vitro. Additionally, students with an interest in gender determination and veterinary medicine can engage in research focused on the XY Sex Reversal Syndrome in the horse as a model for human. Dr. Kent-First earned the MS degree in Anatomy and Cytogenetics and the Ph.D. degree in Physiology and Genetics at the University of Minnesota Graduate School and College of Veterinary Medicine. Dr. Kent-First has published a body of more than 50 peer reviewed articles, 12 Book Chapters, 8 Patents and numerous abstracts and non-peer reviewed publications. She has trained many graduate students and undergraduate students who are currently in successful careers in science.

Early in her graduate career, she made a primary discovery of a unique animal model for mammalian gender determination and gonadal differentiation, namely the XY female horse (XY Sex Reversal Syndrome). Her thesis research utilized this animal model to elucidate the genetic and developmental mechanism by which sexual dimorphism occurs. A professional focus of the Kent-First lab has been aimed at understanding the genetic mechanism by which the gender ambiguous embryo deviates from the default female pathway of development to become a male and is able to produce gametes (in the ovary and the testis). Her work has also led to refined mapping of the Y-chromosome, and a better understanding of mechanisms of mutagenesis associated with reproductive disease including tumorigenesis, genetics, aging and environmental influences on the gonad.

The overall lab focus is on germ line and targeted soma development and tumorigenesis in human and in mouse and animal models. We are interested in the cells response to elevated ROS (oxidative stress) in normal cell development in vivo and in vitro. Our interest encompasses radiation or cadmium induced oxidative stress including understanding the differential responses to high energy and acute damage and low energy damage resulting in delayed response and accumulating mutations and genomic instability that can be heritable. Oxidative stress results in DNA strand breaks that are normally repaired or alternatively the cell dies. Our data suggests that in some cells, oxidative stress simply overrides the cells ability to repair the damage. Regions containing DNA repeats are particularly subject to this damage and when it is left unrepaired in a surviving cell, the mutations in the form of new alleles seen as repeat gains or losses are amplified as cells divide in vitro or in vivo. Thus, genomic instability is a hallmark of oxidative stress and over time, can lead to tumor formation. We have shown that the mature germ cells and precursor stem cells are particularly sensitive to oxidative stress. However, developing cells (stem cells, ovarian stromal cells and primordial germ cells in culture) respond to oxidative stress differently compared with mature “end stage” cells. Specifically, we measure apoptosis and DNA repair in response to ROS and the role that ROS plays in MMR in mature germ cells and somatic cells such as bladder epithelial cells after exposure to ROS. The mechanism of tumor formation is studied. We can measure DNA strand breaks as they occur at the single cell level in parallel to overall chromatin fragility from the progenitor cell and track it over time in culture. Our molecular tools allow us to study the role of ROS induced microsatellite instability in targeted DNA repair genes of functional interest and changes in gene specific methylation."