

**Joint
Nebraska EPSCoR and
Nebraska Gateway for Nutrigenomics
Seminar**

**Dr. Randy Jirtle
Department of Radiation Oncology
Duke University Medical Center**

“Epigenetics, Nutrition, and Disease Susceptibility”

Seminar abstract: Human epidemiological and animal experimental data indicate that the risk of developing adult-onset diseases, such as asthma, diabetes, obesity, and cancer, is influenced by persistent adaptations to prenatal and early postnatal exposure to environmental conditions such as nutritional privation [1]. Moreover, the link between what we are exposed to *in utero* and disease formation in adulthood appears to involve epigenetic mechanisms like DNA methylation at metastable epialleles and imprinted gene loci. Genomic imprinting is an epigenetic form of gene regulation that results in monoallelic, parent-of-origin-dependent gene expression [2]. Since imprinted genes are functionally haploid, only a single genetic or epigenetic event is needed to dysregulate their function. This vulnerability means that imprinted genes are prime candidates for causative roles in human diseases that have a parental inheritance bias and an environmental component in their etiology. We recently developed computer-learning algorithms that predicted the presence of imprinted genes in mice [3] and humans [4]. Not only are humans predicted to have fewer imprinted genes than mice, but there is also a mere 30% overlap between their imprinted gene repertoires. By mapping the human candidate imprinted genes onto the landscape of disease risk defined by linkage analysis, we are now poised to determine the importance of imprinting in the etiology of complex human diseases and neurological disorders.

(The complete abstract with references is available at the NE EPSCoR web site below.)

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4:00 p.m.

**E103 Beadle Center
University of Nebraska-Lincoln**

More information available at
<http://epscor.unl.edu>

