



**Tuesday November 7**

10.00am – 12.30pm

**Poster Session 3**

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Posters P-166 – P-168

## Epigenetics, Chromosomes and Chromatin

**P-166 WILD-DERIVED INBRED MOUSE STRAINS PERA/EI AND PERC/EI POSSESS DOMINANT MODIFIERS THAT RESCUE THE DDK SYNDROME LETHALITY**

F Y Ideraabdullah, K Kim, F Pardo-Manuel de Villena  
University of North Carolina-Chapel Hill, Chapel Hill, NC, United States

**P-167 SEARCHING FOR EPIGENETIC REGULATORY PROTEINS: AN RNAI BASED SCREEN DURING MOUSE PREIMPLANTATION.**

J Mager, M Bartolomei  
University of Pennsylvania, Philadelphia, PA, United States

**P-168 STUDENT ORAL ABSTRACT S-15**

**UTILIZATION OF INBRED MICE TO IDENTIFY THE FIRST *DISTORTER* OF MEIOTIC DRIVE IN METAZOANS.**

HE Doherty, TA Bell, F Pardo-Manuel de Villena  
University of North Carolina - Chapel Hill, Chapel Hill, NC, United States

**P-166****WILD-DERIVED INBRED MOUSE STRAINS PERA/EI AND PERC/EI POSSESS DOMINANT MODIFIERS THAT RESCUE THE DDK SYNDROME LETHALITY**

FY Ideraabdullah, K Kim, F Pardo-Manuel de Villena

University of North Carolina-Chapel Hill, Chapel Hill, NC, United States

The DDK syndrome is an early embryonic lethal phenotype observed in crosses between females of the DDK inbred mouse strain and males of other strains. This phenotype results from an incompatibility between a maternal DDK factor expressed in the oocyte and a non-DDK paternal gene, both of which have been mapped to the *Om* locus on chromosome 11. It has been proposed that the gene encoding the maternal DDK factor is subject to allelic exclusion. Several studies have demonstrated the presence of recessive modifiers of the gene encoding the maternal DDK factor that cause an increase in lethality in crosses involving females that are heterozygous at *Om*. These modifiers are unlinked to *Om* and are thought to be involved in allelic exclusion of the gene encoding the maternal DDK factor such that they skew the choice of which allele is expressed in favor of the DDK allele. Here, we demonstrate the presence of two dominant modifier loci in *Mus musculus domesticus* wild-derived inbred strains, PERA/Ei and PERC/Ei, that are unlinked to *Om* and completely rescue the DDK syndrome lethality. We have mapped one of these modifier loci to the proximal portion of chromosome 13. We hypothesize that PERA/Ei and PERC/Ei dominant modifier alleles influence the choice of allelic exclusion and rescue lethality by increasing the proportion of oocytes that silence the DDK allele of the gene encoding the maternal factor. Ongoing studies will attempt to identify additional modifier loci and verify their involvement in allelic exclusion.

**P-167****SEARCHING FOR EPIGENETIC REGULATORY PROTEINS: AN RNAI BASED SCREEN DURING MOUSE PREIMPLANTATION**

J Mager, M Bartolomei

University of Pennsylvania, Philadelphia, PA, United States

The sequencing of complete genomes allows for comprehensive understanding of the genetic/genomic underpinnings of normal biology and disease states. Uncovering how genomes are epigenetically modified and identification of protein mediators that facilitate epigenetic regulation is a critical next step. We have designed a mammalian RNAi (RNA interference) based screen aimed at identification of novel epigenetic regulatory proteins. Recent advances in mammalian knock-down strategies as well as development of a robust readout of epigenetic regulation have allowed us to design a relatively high throughput epigenetic screen in the mouse. Capitalizing on the dynamic genome reprogramming that occurs during the first few days post-fertilization, our approach uses preimplantation mouse embryos as a model system and both genome imprinting and an Oct4-GFP transgene as epigenetic reporters. To test the feasibility of such a screen, we are first generating lists of candidate genes with dynamic expression patterns during preimplantation with which to perform a pilot screen. To this end, we have merged 3 previously published preimplantation micro-array data sets, and searched for genes that are commonly expressed according to the three data sets. Refinement of these candidates by RT-PCR validation of expression pattern has resulted in lists of genes whose expression patterns are suggestive of a role during preimplantation development. As an early role for most of these genes has not been reported, we hypothesize that our RNAi approach will elucidate previously unknown functions during preimplantation development.

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