**Title of the Contribution:**
A genetic screen for modifiers of the delta1-dependent notch signaling function in the mouse.

**Abstract:**
The Notch signaling pathway is an evolutionarily conserved transduction pathway involved in embryonic patterning and regulation of cell fates during development. Recent studies have demonstrated that this pathway is integral to a complex system of interactions, which are also involved in distinct human diseases. Delta1 is one of the known ligands of the Notch receptors. Mice homozygous for a loss-of-function allele of the Delta1 gene Dll1(lacZ/lacZ) die during embryonic development. Here, we present the results of two phenotype-driven modifier screens. Heterozygous Dll1(lacZ) knockout animals were crossed with ENU-mutagenized mice and screened for dysmorphological, clinical chemical, and immunological variants that are dependent on the Delta1 loss-of-function allele. First, we show that mutagenized heterozygous Dll1(lacZ) offspring have reduced body weight and altered specific clinical chemical parameters, including changes in metabolites and electrolytes relevant for kidney function. In our mutagenesis screen we have successfully generated 35 new mutant lines. Of major interest are 7 mutant lines that exhibit a Dll1(lacZ+)/+ dependent phenotype. These mutant mouse lines provide excellent in vivo tools for studying the role of Notch signaling in kidney and liver function, cholesterol and iron
metabolism, cell-fate decisions, and during maturation of T cells in the immune system.