Docket #: S05-268

The murine ubiquitin B (UbB) gene, (B6;129-Ubbtm1Rrk/J) - JAX 008678

Mice heterozygous for the targeted allele are viable and fertile. This polyubiquitin B (Ubb) mutation is characterized by a GFP-puror fusion protein "knock-in" allele that also abolishes endogenous gene function. Direct visualization of GFP fluorescence is observed in ovaries, testes, hypothalamus (arcuate nucleus) and cerebral cortex. Homozygotes have no Ubb mRNA observed in the various tissues tested, and are viable but sterile due to failure of germ cells to progress through meiotic prophase I and hypogonadism. Homozygotes also exhibit a complex metabolic phenotype initially characterized by dysfunction of neurons within the central nervous system accompanied by retarded perinatal growth that progresses to adult-onset obesity linked to selective hypothalamic neurodegeneration. Homozygotes also develop adult-onset hyperleptinemia (but normal levels of circulating glucose and insulin) as a consequence of increased fat content. These Ubb-mutant mice may be useful in studying gametogenesis, meiosis, neuron development and survival, neuropathogenic disease, and impaired energy balance/metabolism-associated obesity.

Applications

- To study the role of ubiquitin in cellular function.

Advantages

- Currently there are no metazoan models of ubiquitin deficiency. Mice harboring homozygous deletion of UbB (nulls) are viable but are infertile and exhibit an unusual obesity phenotype. These mice may also have reduced life span.

Innovators

- Ron Kopito
- Kwon-Yul Ryu

Licensing Contact

Michael Bellas

Licensing Associate

Email