Docket #: S98-097

Mouse Model for Phosphodiesterase (PDE4D) Deficiency

This mouse model of phosphodiesterase deficiency was developed using homologous recombination to knock-out the gene for PDE4D. The mice have a null PDE4D gene on C57BL/6 x 129/OLA background. These mice have proven useful in studies of asthma (see publications). Also, based on the known function of PDE4, these mice would be useful in studying inflammation, female reproduction, and learning and memory.

Publications

- Lehnart SE, Wehrens XH, Reiken S, Warrier S, Belevych AE, Harvey RD, Richter W, Jin SL, Conti M, Marks AR. "Phosphodiesterase 4D deficiency in the ryanodine-receptor complex promotes heart failure and arrhythmias." *Cell.* 2005 Oct 7;123(1):25-35.
- Nonredundant function of phosphodiesterases 4D and 4B in neutrophil recruitment to the site of inflammation. *J Immunol.* 2004 Dec 15;173(12):7531-8.
- Hansen G, Jin S, Umetsu DT, Conti M., Absence of muscarinic cholinergic airway responses in mice deficient in the cyclic nucleotide phosphodiesterase PDE4D. Proc Natl Acad Sci, 2000 Jun 6;97(12):6751-6.
- S.-L. Catherine Jin, François J. Richard, Wie-Peng Kuo, A. Joseph D'Ercole, and Marco Conti, Impaired growth and fertility of cAMP-specific phosphodiesterase PDE4D-deficient mice, Proc Natl Acad Sci, 1999 Oct 12; 96(21): 11998-12003.

Innovators

- Marco Conti
- Shiow-Lian Jin

Licensing Contact

Brenda Martino

Biological Materials Specialist

Email