Docket #: S21-220

KCNJ2 Knockout strain (Stock No: 005057)

Mice that are homozygous for the targeted mutation have a complete cleft of the secondary palate and die within 12 hours of birth. Heterozygotes are viable, fertile, normal in size and do not display any gross physical or behavioral abnormalities. No gene product (mRNA or protein) is detected by Northern blot analysis of cardiac tissue or Southern blot analysis. Inwardly rectifying potassium ion currents are absent in cerebral artery myocytes and cardiac ventribular myocytes isolated from homozygote neonates. Elevated external potassium ion concentrations do not dilate isolated neonatal cerebral arteries. Homozygotes exhibit altered electrocardiogram profiles indicative of reduced heart rate and bradycardia. This mutant mouse strain may be useful in studies of potassium ion dependent vasodilation, cardiac arrythmia such as Anderson syndrome, cleft palate and developmental bone malformation.

Deposited at Jackson Labs stock number: 005057

Development: A targeting vector containing neomycin resistance and herpes simplex virus thymidine kinase genes was used to disrupt the entire open reading frame. The construct was electroporated into 129-derived R1 embryonic stem (ES) cells. Correctly targeted ES cells were injected into C57BL/6 blastocysts.

Publications

- Targeted disruption of Kir2.1 and Kir2.2 genes reveals the essential role of the inwardly rectifying K(+) current in K(+)-mediated vasodilation.
- Zaritsky JJ , et al.
- PubMed:10904001
- MGI:J:78077
- Circ Res 87(2):160-6

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