

K5R2b Null Mouse

Catalogue number: 151561

Sub-type: Mouse

Images:

Contributor

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Images:

Tool details

***FOR RESEARCH USE ONLY**

Name: K5R2b Null Mouse

Alternate name:

Class:

Conjugate:

Description: A mouse model with epidermal FgfR 2b expression knocked out. The mouse lives a normal life span but has altered hair morphology. It has a tendency to develop skin papillomas spontaneously and development of these can be accelerated greatly by use of a carcinogen.

Purpose:

Parental cell:

Organism:

Tissue:

Model: Knock-Out

Gender:

Isotype:

Reactivity:

Selectivity:

Host:

Immunogen:

Immunogen UNIPROT ID:

Sequence:

Growth properties:

Production details: Homozygous Fgfr2bflox/flox mice, carrying a loxP flanked IIIb exon of FGFR2, were crossed with male heterozygous K5-Cre mice, carrying Cre recombinase transgene under control of the Keratin-5 promoter. Mice were maintained on a mixed 129/C57BL6 background.

Formulation:

Recommended controls:

Bacterial resistance:

Selectable markers:

Additional notes: The K5-R2b-null mouse is an ideal tool in the study of the tumour suppressive role of the FGFR2 IIIb isoform in skin tumourigenesis. Skin tumours develop in K5-R2b-null mice following two-step carcinogen treatment with DMBA and TPA.

Target details

Target: Fibroblast growth factor receptor 2 isoform IIIb (FGFR2IIIb)

Target alternate names:

Target background:

Molecular weight:

Ic50:

Applications

Application: The K5-R2b-null mouse is an ideal tool in the study of the tumour suppressive role of the FGFR2 IIIb isoform in skin tumourigenesis. Skin tumours develop in K5-R2b-null mice following two-step carcinogen treatment with DMBA and TPA.

Application notes:

Handling

Format:

Concentration:

Passage number:

Growth medium:

Temperature:

Atmosphere:

Volume:

Storage medium:

Storage buffer:

Storage conditions:

Shipping conditions: Embryo/Spermatozoa- Dry Ice

Related tools

Related tools:

References

References: Stringer et al. 2012. Development. 139(3):465-74. PMID: 22190642. ; Cdx2 determines the fate of postnatal intestinal endoderm. ; Kemp et al. 2004. Nucleic Acids Res. 32(11):e92. PMID: 15247325. ; Elimination of background recombination: somatic induction of Cre by combined transcriptional regulation and hormone binding affinity. ; Ireland et al. 2004. Gastroenterology. 126(5):1236-46. PMID: 15131783. ; Inducible Cre-mediated control of gene expression in the murine gastrointestinal tract: effect of loss of beta-catenin.

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