

# PI3K-C2b kinase-dead

**Catalogue number:** 158398

**Sub-type:** Mouse

**Images:**

## Contributor

**Inventor:** Bart Vanhaesebroeck

**Institute:** Ludwig Institute for Cancer Research

**Images:**

## Tool details

**\*FOR RESEARCH USE ONLY**

**Name:** PI3K-C2b kinase-dead

**Alternate name:**

**Class:**

**Conjugate:**

**Description:**

**Purpose:**

**Parental cell:**

**Organism:**

**Tissue:**

**Model:** Knock-In

**Gender:**

**Isotype:**

**Reactivity:**

**Selectivity:**

**Host:**

**Immunogen:**

**Immunogen UNIPROT ID:**

**Sequence:**

**Growth properties:**

**Production details:** Knock-in mice in which the endogenous PIK3C2B/PI3K-C2beta PI3K gene is mutated so that it now encodes a PI3K-C2beta protein with the D1212A mutation in the ATP binding site, converting it to a kinase-dead PI3K-C2beta protein which is expressed at the same level as wild-type PI3K-C2beta. These mice have been backcrossed onto the B6 background.

**Formulation:**

**Recommended controls:**

**Bacterial resistance:**

**Selectable markers:**

**Additional notes:** Homozygous mice are phenotypically normal and born at a normal Mendelian ratio, with no impact on organismal growth. Mice display enhanced insulin sensitivity and glucose tolerance, as well as protection against high-fat-diet-induced liver steatosis. (see PMID 26655903 for details). Heterozygous mice are phenotypically normal.

## Target details

**Target:** PIK3C2B

**Target alternate names:**

**Target background:**

**Molecular weight:**

**Ic50:**

## Applications

**Application:**

**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:** Valet et al. 2015. Blood. 126(9):1128-37. PMID: 26109204.

CancerTools.org