B6/JGpt-Tek-P2A-CreERT2

Strain Name: C57BL/6JGpt-*Tek*$^{em1Cin(P2A-CreERT2)}$/Gpt
Strain Type: Knock-in
Strain ID: T069086
Background: C57BL/6JGpt

Description

This mouse strain expresses CreERT2 inducible recombinase [1] under the control of the mouse *Tek* promoter, P2A-CreERT2 was precisely inserted downstream of TAG by CRISPR/Cas9 technology. When crossed with a strain with loxP site flanked sequence in its genome, Cre-mediated recombination will result in excision of the DNA fragment between the two loxPs in endothelial cells and hematopoietic cells after tamoxifen administration.

Strategy

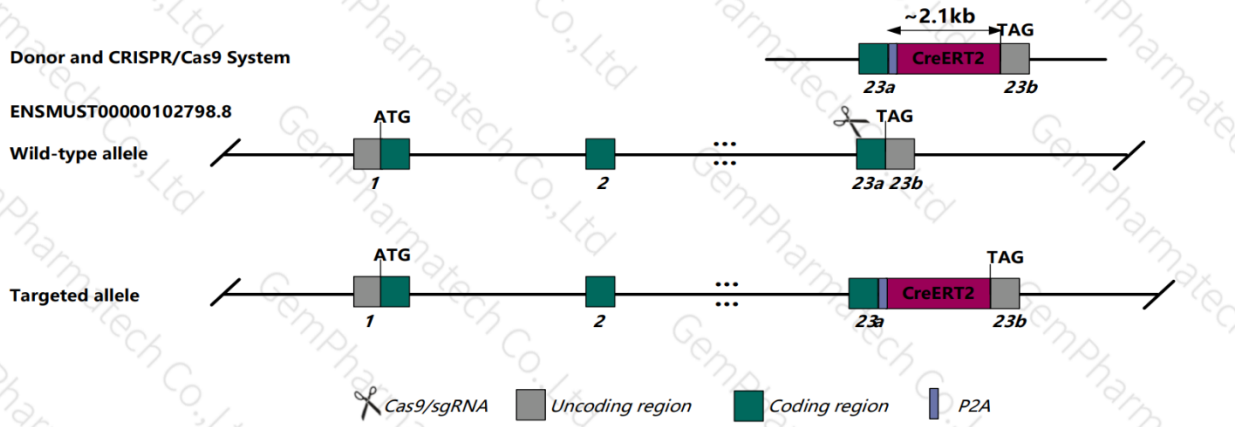


Fig 1. Schematic diagram of B6/JGpt-Tek-P2A-CreERT2model strategy

Applications

1. Cre tool mice for specific, tamoxifen dependent induction of loxP recombination in endothelial cells and hematopoietic cells [2-3].

Data support

1. Validation methods

Tek-P2A-CreERT2 mice were crossed with Rosa26-loxp-ZsGreen-Stop-loxp-tdTomato mice with ubiquitous reporter expression (hereafter referred as CAG-G/R mice), Cre-mediated

recombination leads to excision of ZsGreen and the stop cassette and expression of tdTomato, thus loss of green fluorescence and gain of red fluorescence indicates Cre activity. Fluorescence imaging of frozen sections was performed to exhibit Cre activity in various tissues and organs. Imaging sections were performed under a 200x microscopy. For tamoxifen administration, 100 mg/kg tamoxifen was treated through intraperitoneal injection daily from P44 to P50 (6.3 w~7.1 w).

Note: these results may only represent the activity of CreERT2 in this strain under this certain tamoxifen treatment condition at the identical stage. Recombinase activity may be different at other stages or under different tamoxifen induction conditions in your application.

Tek-P2A-CreERT2 mice exhibited CreERT2 activity in the heart, liver, spleen, lung, kidneys, small intestine, skeletal muscles, aorta, brain, with little or no CreERT2 activity in the sperm after tamoxifen administration. Sperm from Tek-P2A-CreERT2 mice exhibited no CreERT2 activity prior to tamoxifen administration. It should be noted that other tissues remain untested, and the presence of CreERT2 in unexamined organs is not guaranteed.

2. Timeline of tamoxifen treatment and imaging

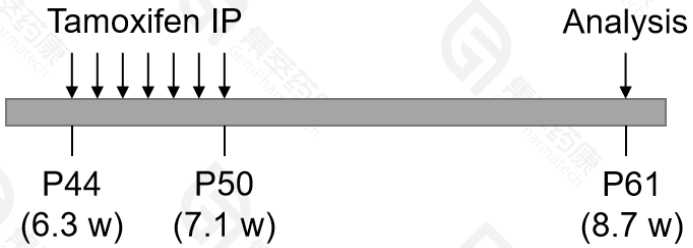
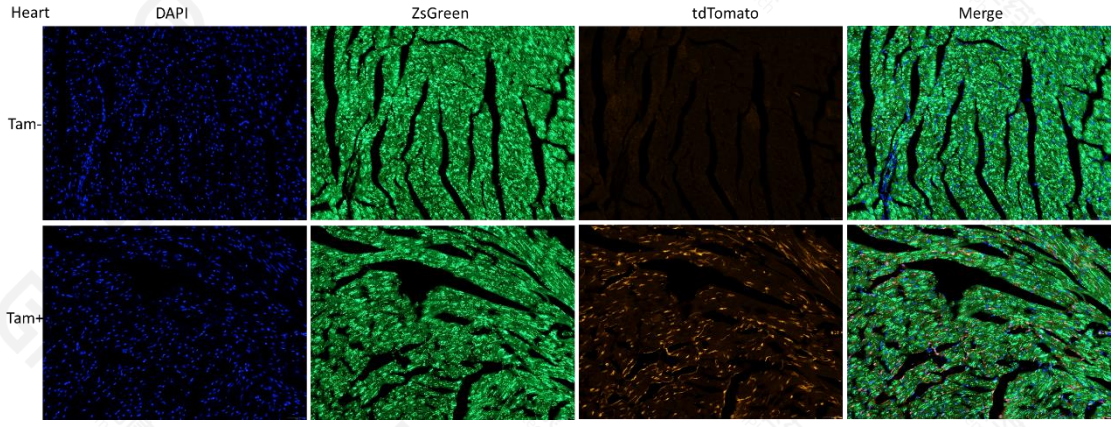
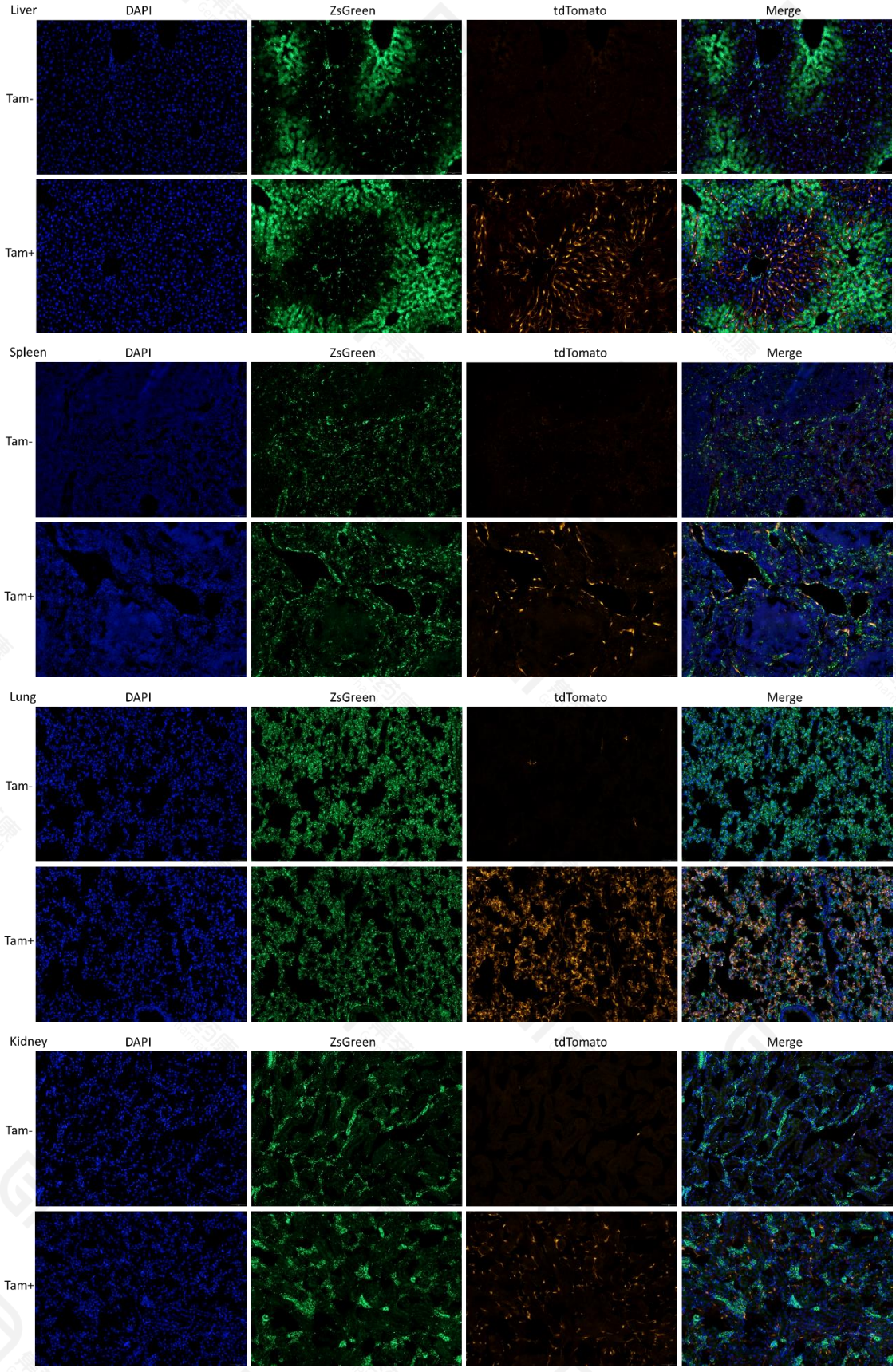


Fig 2. Timeline of tamoxifen treatment and experiment analysis of Tek-P2A-CreERT2 mice.

3. Images of tissues and organs with obvious Cre activity




Liver
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+
Spleen
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+
Lung
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+
Kidney
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+

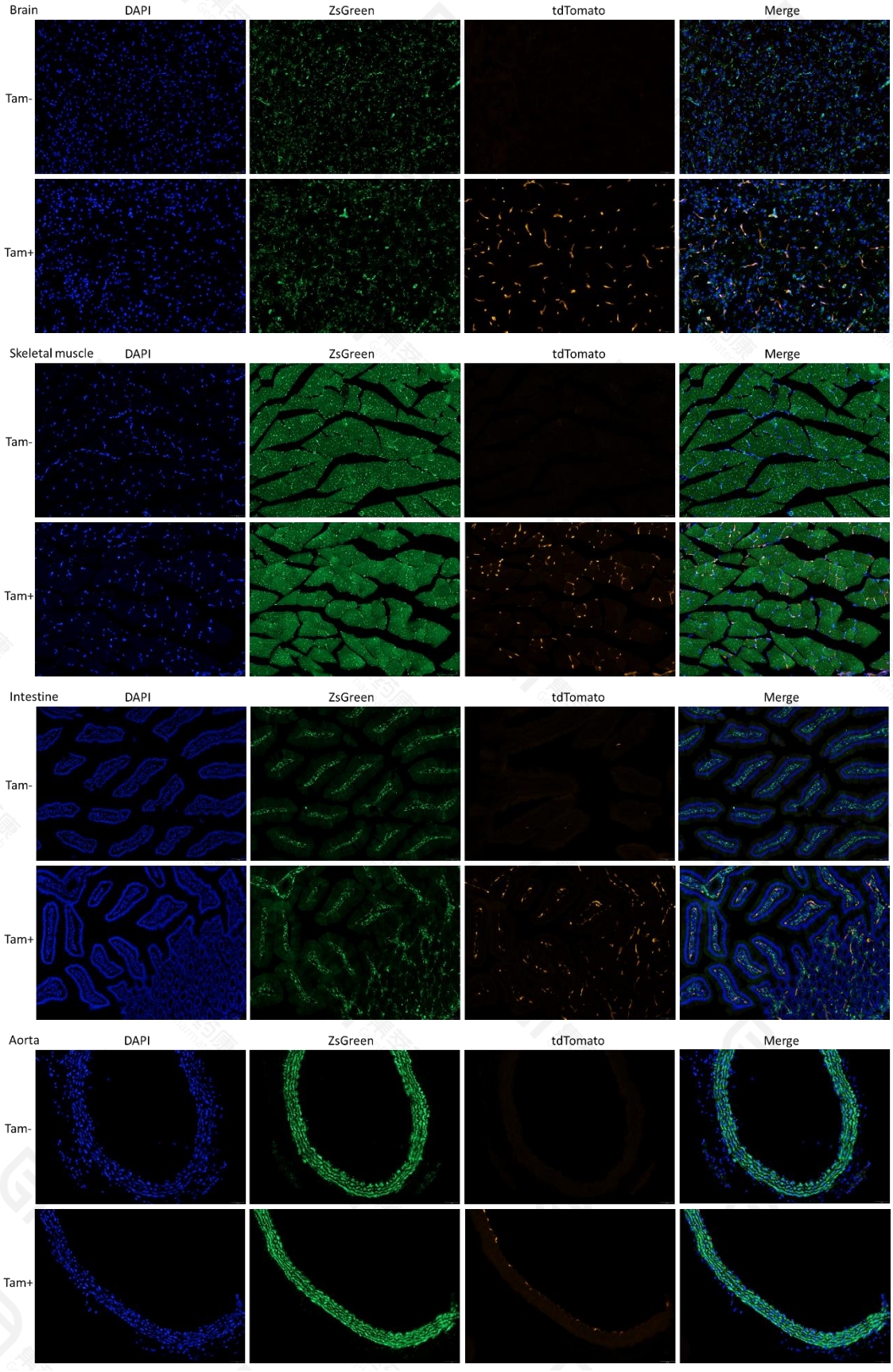

Brain
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+
Skeletal muscle
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+
Intestine
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+
Aorta
DAPI
ZsGreen
tdTomato
Merge
Tam-
Tam+

Fig 3. Fluorescence imaging of tissues and organs with obvious Cre activity.

Organ name was indicated in the left top of each subfigure group. Tam-: Tek-P2A-CreERT2, CAG-G/R double positive individuals without tamoxifen administration; Tam+: Tek-P2A-CreERT2, CAG-G/R double positive individuals with tamoxifen administration.

Reference

1. Feil, Robert, et al. "Regulation of Cre recombinase activity by mutated estrogen receptor ligand-binding domains." *Biochemical and biophysical research communications* 237.3 (1997): 752-757.
2. Forde, Anne, et al. "Temporal Cre-mediated recombination exclusively in endothelial cells using Tie2 regulatory elements." *genesis* 33.4 (2002): 191-197.
3. Tang, Yuefeng, et al. "The contribution of the Tie2+ lineage to primitive and definitive hematopoietic cells." *genesis* 48.9 (2010): 563-567.