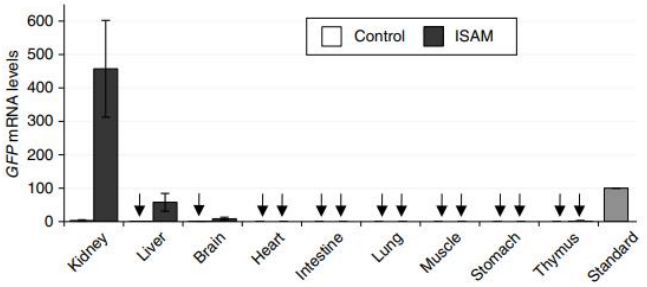
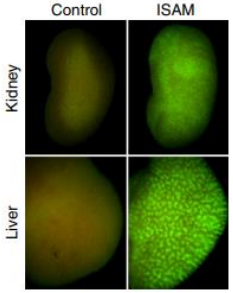
Tohoku Univ. Technology

# Epo-deficient GFP anemic mice

## Anemia developed after birth without fetal lethality




## Overview

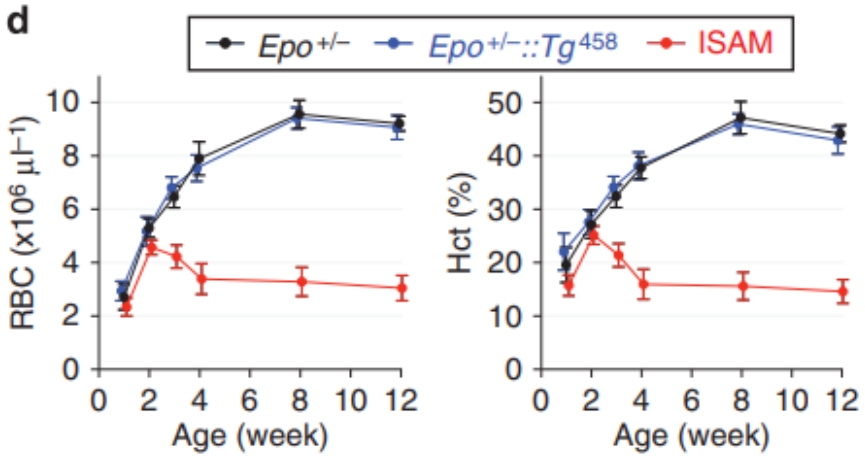
Although the development of erythropoietin (Epo) -related drugs requires the existence of animal models for disease states such as renal anemia, Epo knockout mice are not suitable as a model because of the embryonic lethality and the large inter-experiment/inter-individual differences in the anemia state in the models produced by surgery/drug treatment. The present invention relates to transgenic mice (I-SAM) that have overcome these problems.

In I-SAM, the endogenous Epo gene is homologically knocked out by insertion of the GFP gene, and a region from 3.3kbp upstream to 4.5kbp downstream of the Epo gene transcription start point (Transgene) is introduced. In I-SAM before birth, transgene-derived Epo is expressed in the liver, etc., and there is no embryonic lethality. After birth, transgene does not express Epo because it has no transcriptional activity in the major Epo-producing tissues, such as the kidney, and there is anemia.

I-SAM has the following characteristics.

- Epo knockout but not embryonic lethality
- Stably develop high levels of anemia after birth
- Have normal renal function
- Have tissue Epo-producing cells labeled with GFP
- Have a long lifespan and can reproduce by mating

## Anemic manifestations of I-SAM occur after birth




## Possible applications

- Evaluation test after eliminating the effect of endogenous Epo
- Evaluation the effects of the drug candidates in anemia drugs' discovery research and development

## Related Works

Yamazaki S, et al : A mouse model of adult-onset anaemia due to erythropoietin deficiency. Nat Commun 4 : 1950, 2013

## IP Data

IP No. : JP5939487
Inventor : YAMAMOTO Masayuki, MINEGISHI Naoko, YAMAZAKI Shun
Admin No. : T10-050

## Contact

**Tohoku Techno Arch Co., Ltd.**
Please visit CONTACT here