Prime Scholars Library

Advances in Animal Science, Theriogenology, Genetics and Breeding

Vol. 10 (3), pp.61, December, 2022

Perspective

Available online at
https://primescholarslibrary.org/

Transgenic mouse and its model in genetic breeding

Oliva Liam*

Department of Animal Science, University of Oxford, Oxford, USA

Received: 25-Nov-2022, Manuscript No. AASTGB-22-88053; **Editor assigned:** 28-Nov-2022, PreQC No. AASTGB-22-88053(PQ); **Reviewed:** 15-Dec-2022, QC No AASTGB-22-88053; **Revised:** 22-Dec-2022, Manuscript No. AASTGB-22-88053 (R); **Published:** 29-Dec-2022, DOI: 10.51268/2736-1810-22.10.061

DESCRIPTION

In the absence of a natural animal model for sickle cell disease, transgenic mice models have been developed to better understand the illness's complex biology and to evaluate potential particular treatments. In the early 1990s, the simple insertion of human globin genes into mice still expressing mouse hemoglobin resulted in the development of hemoglobin S (HbS) or HbS-related human hemoglobin.

A combination of mouse alpha and defects might be utilized to reduce the percentage of mouse haemoglobin resulting in complex genotype and moderate sickness, to increase the proportion of human haemoglobin and the severity of the mouse sickle cell syndrome. It was possible to delete all adult mouse globin genes (2alpha and 2beta) and replace them with human equivalent genes elsewhere in the mouse genome immediately following the discovery of gene targeting in mouse Embryonic Stem Cells (ES cells). Furthermore, the prenatal haemoglobin human gamma gene shielded the foetus from the development of HbS polymers. As a result adult mouse models that only expressed human HbS were established in 1997.

Adding modified human gamma genes, which are still expressed in adult mice could help to alleviate the condition. In 2006, a final "S-only" model was created using homologous knock in, with human genes replacing mouse globin genes. This collection of models aids researchers in better understanding the role of various interacting factors in sickle cell complications such as red cell defects, blood flow and vaso-occlusion, hyperhemolysishemolysis, capillary tone marketization, oxidations, inflammation, cell activation and adhesion, ischemia and reperfusion. Furthermore, each animal is ideal for testing new drugs.

Researchers developed beta SAD, a novel human beta-globin gene that promotes the polymerization of transgenic human haemoglobin S (Hb S) to construct a transgenic mouse model of sickle cell disease. The beta 6Val beta S chain mutation, as well as two other variants, Antilles (beta 23Ile) and D Punjab (beta 121Gln), all increase Hb S polymerization in humans. Both the beta SAD and human alpha 2-globin genes were inserted into the mouse germ line via the beta-globin locus regulatory area (LCR). SAD-1, one of the five transgenic lines obtained, has 19% human Hb SAD (alpha 2 human 1 beta 2SAD) and mouse-human hybrids in its red blood cells, in addition to mouse haemoglobin.

Adult SAD-1 transgenic mice were not anaemic, but their erythrocytes were abnormal, and their livers were somewhat enlarged. Deoxygenation in produced sickling in their erythrocytes. Many

SAD-1 newborns perished as a result of anemia. To create adult mice with a more severe sickle cell disease, researchers crossed SAD progeny with homozygous for beta-thalassemia animals.

Sickle cell disease is a genetic condition characterised by red blood cell distortion caused by haemoglobin polymerization. The possible healing threshold of foetal haemoglobin in a transgenic SAD mouse model of sickle cell disease was tested by mating with mice expressing the human foetal agama globin gene. Agamas mice demonstrated significant improvements in all haematological parameters, morphological pathological characteristics, and longevity or survival with higher HbF levels. In this mouse model, we established a direct therapeutic effect on foetal haemoglobin for sickle cell disease and proved correction by boosting foetal haemoglobin to nearly 916%.