

Resource Summary Report

Generated by ASWG on Aug 3, 2026

Induced Mutant Resource

RRID:SCR_008366

Type: Tool

Proper Citation

Induced Mutant Resource (RRID:SCR_008366)

Resource Information

URL: <http://www.jax.org/imr/index.html>

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Description: THIS RESOURCE IS NO LONGER IN SERVICE, documented on June 08, 2012. The function of the IMR is to select, import, cryopreserve, maintain, and distribute these important strains of mice to the research community. To improve their value for research, the IMR also undertakes genetic development of stocks, such as transferring mutant genes or transgenes to defined genetic backgrounds and combining transgenes and/or targeted mutations to create new mouse models for research. The function of the IMR is to: * select biomedically important stocks of transgenic, chemically induced, and targeted mutant mice * import these stocks into the Jackson Laboratory by rederivation procedures that rid them of any pathogens they might carry * cryopreserve embryos from these stocks to protect them against accidental loss and genetic contamination * backcross the mutation onto an inbred strain, if necessary * distribute them to the scientific community More than 1000 mutant stocks have been accepted by the IMR from 1992 through December 2006. Current holdings include models for research on cancer; breast cancer; immunological and inflammatory diseases; neurological diseases; behavioral, cardiovascular and heart diseases; developmental, metabolic and other diseases; reporter (e.g., GFP) and recombinase (e.g., cre/loxP) strains. About eight strains a month are being added to the IMR holdings. Research is being conducted on improved methods for assisted reproduction and speed congenic production. Most of the targeted mutants arrive on a mixed 129xC57BL/6 genetic background, and as many of these as possible are backcrossed onto an inbred strain (usually C57BL/6J). In addition, new mouse models are being created by intercrossing carriers of specific transgenes and/or targeted mutations. Simple sequence length polymorphism DNA markers are being used to characterize and evaluate differences between inbred strains, substrains, and embryonic stem cell lines.

Abbreviations: IMR

Resource Type: organism supplier, material resource, biomaterial supply resource

Keywords: embryo, genetic, behavioral, biomedical, breast cancer, cancer, cardiovascular, chemical, cre, cryopreserved, developmental, disease, distribution, dna, gfp, heart, immunological, inflammatory, loxp, marker, metabolic, model, mouse, mutation, neurological, pathogen, polymorphism, recombinase, research, stock, targeting, transgene

Funding: March of Dimes Birth Defects Foundation ;
American Cancer Society ;
American Heart Association ;
Cystic Fibrosis Foundation ;
National Multiple Sclerosis Society ;
Amyotrophic Lateral Sclerosis Association ;
NIAID ;
NIAMS ;
Howard Hughes Medical Institute ;
Department of the Army Breast Cancer Research Initiative. ;
NCRR P40 RR009781

Availability: THIS RESOURCE IS NO LONGER IN SERVICE

Resource Name: Induced Mutant Resource

Resource ID: SCR_008366

Alternate IDs: nif-0000-25566

Record Creation Time: 20220129T080247+0000

Record Last Update: 20260803T040523+0000

Ratings and Alerts

No rating or validation information has been found for Induced Mutant Resource.

No alerts have been found for Induced Mutant Resource.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 3 mentions in open access literature.

Listed below are recent publications. The full list is available at [ASWG](#) .

Ferrer-Vaquero A, et al. (2010) A sensitive and bright single-cell resolution live imaging reporter of Wnt/??-catenin signaling in the mouse. BMC developmental biology, 10, 121.

Long JZ, et al. (2005) Genetic and spectrally distinct in vivo imaging: embryonic stem cells and mice with widespread expression of a monomeric red fluorescent protein. BMC

biotechnology, 5, 20.

Hadjantonakis AK, et al. (2004) Dynamic in vivo imaging and cell tracking using a histone fluorescent protein fusion in mice. BMC biotechnology, 4, 33.