

charles river
accelerating drug development. exactly.

Oncology Animal Models

The following information provides an overview of Charles River's portfolio of immunodeficient and immunocompetent oncology animal models.

Immunodeficient Animal Models

Immunodeficient animal models are extremely useful in a wide range of biomedical research, including infectious disease, stem cell, immunology and oncology studies. Due to the unique vulnerability that makes these models vital to research, their care and maintenance demands a high level of expertise and technological resources.

Strain	Hair	T-Cell Deficient	B-Cell Deficient	NK Cell Deficient
Athymic Nude Mouse	No	Yes	No	No
CD-1® Nude Mouse	No	Yes	No	No
Swiss Nude Mouse	No	Yes	No	No
BALB/c Nude Mouse	No	Yes	No	No
NMRI Nude Mouse	Yes	Yes	No	No
NIH-III Mouse	No	Yes	Yes	Impaired
RNU Rat	No	Yes	No	No
SCID Hairless Outbred (SHO®) Mouse	No	Yes	Yes	No
SCID Hairless Congenic (SHC™) Mouse	No	Yes	Yes	No
Fox Chase SCID® Congenic Mouse	Yes	Yes	Yes	No
Fox Chase SCID® Beige Mouse	Yes	Yes	Yes	Impaired
NOD SCID Mouse	Yes	Yes	Yes	Impaired
NOD *scid* gamma (NSG) Mouse	Yes	Yes	Yes	Yes

Nude Models

Athymic Nude Mouse
Nomenclature: Crl:NU(NCr)-*Foxn1*^{nu} Origin: This immunodeficient nude mouse originated from NIH and was originally thought to be a BALB/c congenic. It was later determined that it was not inbred and is, therefore, maintained as an outbred. This model is not associated with any stock or strain. The animal lacks a thymus, is unable to produce T cells, and is therefore immunodeficient. To Charles River from NCI in 2010.

Overview

Oncology is one of the leading areas of research into new therapeutics. Due to the challenges inherent in researching and developing anticancer therapeutics, it is critical that you have the right tools and resources available to you. Backed by decades of technical, scientific and veterinary experience, Charles River's global portfolio of high-quality oncology models gives you the benefit of partnering with an industry leader offering an infrastructure capable of advancing your research now and in the future.



CD-1® Nude Mouse

Nomenclature: Crl:CD1-*Foxn1*^{nu} Origin: Developed from the transfer of the nude gene from Crl:NU-*Foxn1*^{nu} to a CD-1 mouse through a series of crosses and backcrosses beginning in 1979 at Charles River Wilmington, MA. The animal lacks a thymus, is unable to produce T cells and is therefore immunodeficient.

Swiss Nude Mouse

Nomenclature: Crl:NU(Ico)-*Foxn1*^{nu} Origin: These nude mice originate from the Swiss stock. In 1974, the central animal breeding facilities of Gustave Roussy Institute, Villejuif, France, obtained these mice from Dr. Carl Hansen's department at the NIH. The first pairs were introduced to Charles River, France, in 1976. Characteristics: T-cell deficient. Hairless; albino

BALB/c Nude Mouse

Nomenclature: CAnN.Cg-*Foxn1*^{nu}/Crl Origin: Developed through crosses and backcrosses between BALB/cABom-nu and BALB/cAnNCrj-nu at Charles River Japan. Pedigreed pregnant females of CAnN.Cg-*Foxn1*^{nu}/Crl were received from Charles River Japan in 1985. This mouse is inbred, and genetic monitoring results confirm it to be a BALB/c nude. The homozygous animal lacks a thymus, is unable to produce T cells and is therefore immunodeficient.

BALB/c Nude Mouse - JAX® Mice strain

Nomenclature: CBy.Cg-*Foxn1*^{nu}/J JAX® Mice Stock number: 000711 Origin: The strain BALB/cByJ-*Foxn1*^{nu} was created by repeated backcrosses to move the nude gene onto the BALB/cByJ background. In September 1986, the first BALB/cByJ nu/+ pairs were introduced into Charles River France, from The Jackson Laboratory, Bar Harbor, Maine, USA. Characteristics: T-cell deficient. Hairless; albino.

Ask for the J

NIH-III Mouse

Nomenclature: Crl:NIH-*Lyst*^{bg}*Foxn1*^{nu}*Btk*^{xid} Origin: Most commonly called the NIH-III, it was developed at NIH. In addition to the nude gene, which results in the absence of thymus and T-cell function, this mouse has two other mutations important in regulating the function of the immune system. These are designated as x-linked immune defect (*xid*) and beige (*bg*). The *xid* mutation affects the maturation of T-independent B lymphocytes. It has been demonstrated that *bg* homozygotes have defective natural killer (NK) cells that are cytotoxic *in vitro* to tumor cells. However, the extent of the T-independent B-lymphocyte and NK cell deficiencies in the NIH-III have not been established.

NMRI Nude Mouse

Nomenclature: Crl:NMRI-*Foxn1*^{nu} Origin: Developed from the transfer of the nude gene to a NMRI mouse through a series of outcrosses. Characteristics: The model is T-cell deficient. Outbred; albino.

RNU Nude Rat

Nomenclature: Crl:NIH-*Foxn1*^{rnu} Origin: The NIH nude rat was developed in 1979–1980 through a series of matings involving eight inbred rat strains. To Charles River from the National Institutes of Health in 2001. This athymic nude rat is T-cell deficient and shows depleted cell populations in thymus-dependent areas of peripheral lymphoid organs.

Severe Combined Immunodeficiency (SCID) Models

SCID Hairless Outbred (SHO®) Mouse

Nomenclature: Crl:SHO-*Prkdc*^{scid}*Hr*^{hr} Origin: The hairless SCID mouse was produced by Charles River Research Models in 2007 by intercrossing the Crl:HA-*Prkdc*^{scid} and Crl:SKH1-*Hr*^{hr} stocks. The resulting animals are homozygous for the *Prkdc*^{scid} and the *Hr*^{hr} mutations and thus exhibit the severe combined immunodeficiency phenotype characteristic of SCID mice and are also hairless.

SCID Hairless Congenic (SHC™) Mouse

Nomenclature: CB17.Cg-*Prkdc*^{scid}*Hr*^{hr}/IcrCrl Origin: The hairless SCID congenic was created in 2009 by Charles River Research Models by using marker-assisted accelerated backcrossing to place the hairless gene (*Hr*^{hr}) present in the Crl:SKH1-*Hr*^{hr} stock onto a CB17/Icr-*Prkdc*^{scid}/IcrIcoCrl genetic background. These mice are homozygous for both *Hr*^{hr} and *Prkdc*^{scid} mutations, so exhibit the severe combined immunodeficiency phenotype characteristic of SCID mice and are also hairless.

Fox Chase SCID® Congenic Mouse

Nomenclature: CB17/Icr-*Prkdc*^{scid}/IcrIcoCrl Origin: SCID mice possess a genetic autosomal recessive mutation (SCID). Discovered in 1980 by Bosma in C.B-17/Icr mice at Fox Chase Cancer Center. SCID mice show a severe combined immunodeficiency affecting both B and T lymphocytes. They have normal natural killer (NK) cells, macrophages, and granulocytes. To Charles River in 1991 from a Charles River France foundation colony.

Fox Chase SCID® Beige Mouse

Nomenclature: CB17.Cg-*Prkdc*^{scid}*Lyst*^{bg}/Crl Origin: A congenic mouse that possesses both autosomal recessive mutations SCID (*Prkdc*^{scid}) and beige (*Lyst*^{bg}). The SCID mutation results in severe combined immunodeficiency affecting both the B and T lymphocytes. The beige mutation results in defective natural killer (NK) cells. This mouse was developed by Croy, *et al.*, at the University of Guelph by an intercross of C.B-17 *scid*/*scid* to C57BL/6 *bg*/*bg* mice. To Charles River in 1993.

NOD SCID Mouse

Nomenclature: NOD.CB17-*Prkdc*^{scid}/NcrCrl Origin: The SCID mutation has been transferred onto a non-obese diabetic background. Animals homozygous for the SCID mutation have impaired T- and B-cell lymphocyte development and impaired natural killer (NK) cells. To Charles River in 2005 from Frederick Cancer Research and Development Center.

NOD SCID Mouse - JAX® Mice strain

Ask for the J

Nomenclature: NOD.CB17-*Prkdc*^{scid}/J JAX® Mice Stock number: 001303 Origin: Spontaneous mutation congenic on the NOD/ShiLtJ background. Mice are homozygous for the scid mutation (*Prkdc*^{scid}). Mutation occurs in the gene encoding the catalytic sub unit of DNA activated protein kinase (*Prkdc*). To Charles River France from The Jackson Laboratory in March 2003. Characteristics: B and T cell deficient. NK cells impaired. Inbred; albino.

NOD *scid* gamma (NSG) Mouse - JAX® Mice strain

Ask for the J

Nomenclature: NOD.Cg-*Prkdc*^{scid}*Il2rg*^{tm1Wjl}/SzJ JAX® Mice Stock number: 005557
Origin: These mutant mice combine the features of the NOD/ShiLtJ background which confers a number of deficiencies in innate immunity; the severe combined immune deficiency mutation (*scid*) and also IL2 receptor gamma chain deficiency due to gene targeting. To Charles River UK from The Jackson Laboratory in 2010. Characteristics: B and T cell deficient, without leakiness. Lacks the gene IL2R-g (gamma c) - which is a key immune signalling molecule. Does not produce detectable serum immunoglobulin. No NK cell activity. Engrafts the widest variety of human cells, tissues, and cancers. Inbred, albino.

Other JAX® Mice strains for oncology research are available as imported models.
Charles River can coordinate all importation steps.

Only The Jackson Laboratory and Charles River in Europe and Japan maintain colonies of JAX® Mice strains which are derived from pedigreed mice from The Jackson Laboratory and are re-infused routinely with pedigreed mice to stabilise the genetic integrity and phenotypes of these strains. Authentic JAX® Mice strains are designated by a 'J' as the final character in the strain name. JAX® Mice are for internal research use only and should not be propagated for distribution or sale.

Immunocompetent Models

Buffalo Rat

Nomenclature: BUF/CrCrl Origin: Heston in 1946 from Buffalo stock of H. Morris. To NIH in 1951 at F10. To Charles River in 1998 from the National Cancer Institute Animal Production Program (Cr). Coat Color: White (Albino).

p53 TGEM® Knockout Rat

Nomenclature: Crl:WI(UL)-*Tp53*$^{tm1/Hubr}$ Origin: At the Hubrecht Institute, a nonsense mutation at amino acid position 273 (Cys to stop) within the DNA binding domain of the rat p53 gene was isolated, which resulted in a full knockout mutation. Systematic generation of the TGEM® knockout rats was carried out by random mutagenesis of Wistar rats followed by PCR amplification and capillary sequencing. Transferred to Charles River under exclusive license from Transposagen in 2010. Coat Color: White (Albino).

Wistar Furth Rat

Nomenclature: WF/IcoCrl Origin: Furth developed this strain at Roswell Park Memorial Institute, Buffalo, NY, USA in 1945 starting from a commercial colony of Wistar rats. Acquired by Charles River from Microbiological Associates, Bethesda, Maryland, USA. Introduced to Charles River France in 1970. Coat color: White (Albino).

Immortomouse®*

Nomenclature: CBA;B10-Tg(H2K^{b}-tsA58)6Kio/Crl Origin: At the Ludwig Institute for Cancer Research, a hybrid construct containing H-2K^{b} (MHC Class I antigen) 5' promotor sequences fused to the early region of the SV40 mutant tsA58, which encodes both the large and small SV40 tumor antigens, was microinjected into fertilized oocytes from CBA/Ca x C57BL/10 F1 mice. Following reimplantation, 88 mice were born, of which 34 were transgenic and carried one to five copies of the gene. RNA from a variety of tissues from one nontransgenic and three transgenic animals was analyzed by Northern blot analysis using a SV40 early region-specific probe. RNA extracted from tissues of transgenic mice contained varying amounts of a 2.5kb RNA species, while no tsA58 TAg RNA was detected in tissues of the nontransgenic mouse; thymus and liver showed the highest level of expression, while brain showed the lowest. Distribution rights to Charles River in 1991. Coat Color: Primarily agouti, infrequently black.

**Transgenic, isolator-maintained*

Cryopreserved Oncology Models

The strains listed below are currently maintained as frozen embryos. Two breeding pairs can be made available upon request. Please contact Customer Service for pricing and availability.

Common Name	Nomenclature	Species	Coat Color
AKR	AKR/NCrl	Mouse	White (Albino)
BDIX	BDIX/CrCrl	Rat	Agouti
Noble	NBL/CrCrl	Rat	White with black face/hood
Wistar Furth	WF/CrCrl	Rat	Albino
B6 D933A	B6J.129P2-*Pik3ca*^{tm2Bvan}/Crl	Mouse	Black
p110alpha flox	B6J.129P2-*Pik3ca*^{tm1Bvan}/Crl	Mouse	Black
B6 LacZ	B6J.129P2-*Pik3ca*^{tm3Bvan}/Crl	Mouse	Black
p110beta flox	B6J.129P2-*Pik3cb*^{tm1Bvan}/Crl	Mouse	Black
B6 lacZ D931A	B6J.129P2-*Pik3cb*^{tm2Bvan}/Crl	Mouse	Black
B6 D931A	B6J.129P2-*Pik3cb*^{tm3Bvan}/Crl	Mouse	Black
129 D931A	129P2.B6J-*Pik3cb*^{tm3Bvan}/Crl	Mouse	Agouti
C D931A	C.129P2(B6)-*Pik3cb*^{tm3Bvan}/Crl	Mouse	White (Albino)
B6 flox	B6J.129P2-*Pik3cb*$^{tm1.1Bvan}$/Crl	Mouse	Black
129 flox	129P2;B6J-*Pik3cb*$^{tm1.1Bvan}$/Crl	Mouse	Agouti
lacZ	B6J.129P2-*Pik3cd*^{tm1Bvan}/Crl	Mouse	Black
B6D910A	B6J.129P2-*Pik3cd*^{tm2Bvan}/Crl	Mouse	Black
C D910A	C.129P2(B6)-*Pik3cd*^{tm2Bvan}/Crl	Mouse	Agouti
B6 S1031A	B6J.129P2-*Pik3cd*^{tm3Bvan}/Crl	Mouse	Black

Contact Us
askcharlesriver@crl.com
www.criver.com