



Immunodeficient Models

The breeding of immunodeficient models is a highly specialized task. Charles River has combined technological advances made in the production of these important research models with the knowledge and experience of our staff to become experts in breeding immunodeficient models.

Health monitoring is stringent, and animals are maintained with a SOPF health status (Specific and Opportunistic Pathogen Free). Some of our procedures to ensure the health status of these animals include:

- Sterile feed and bedding;
- Housing the animals in isolators or filter top cages;
- Weekly microbiological testing of faecal pools;
- Monthly serological testing of immunocompetent sentinels or colony animals;
- Full health monitoring every quarter within each breeding unit;
- Including a health report with every immunodeficient model order.

Animals also receive irradiated feed as their shipping diet. From their conception to their delivery, our customers can rest assured that immunodeficient research models supplied by Charles River undergo the most stringent, effective production processes possible.

What makes a model immunodeficient?

Athymic - Mutation

- *Foxn1^{nu}* gene spontaneous mutation
- Thymic anlage dysgenesis
- T cell deficiency

SCID - Mutation

- *Prkdc^{scid}* (protein kinase, DNA activated catalytic polypeptide) gene spontaneous mutation
- Defects in DNA repair which leads to defects in late phase VDJ (Variable diversity joining) recombination in the lymphocyte
- B and T cell deficiency

Beige - Mutation

- *Lyst^{tg}* (lysosomal trafficking regulator) gene spontaneous mutation
- Natural Killer cell defect
- Beige mutation mice block the normal process of degranulation leading to impaired antibody-dependent and antibody-independent cytotoxicity of tumor cells.

XID - Mutation

- *Btk^{xid}* (bruton's tyrosine kinase) gene spontaneous mutation
- X chromosome-linked immune defect
- B cells are produced, but are functionally immature and abnormal in the production of antibodies

Research Applications

- Xenograft tumor studies
 - Efficacy
 - Toxicity
- Infectious disease research
- Lymphocyte stem cell research
- Immunology
- Ascites production for monoclonal antibody



Immunodeficient Models

CD-1 Nude Mouse - Nomenclature: Crl:CD1-Foxn1^{nu}

Origin: Developed from the transfer of the nude gene to a CD-1 mouse through a series of outcrosses and back-crosses, beginning in 1979 at Charles River, Wilmington, MA.

Characteristics: T-cell deficient. Hairless; albino.

Swiss Nude - Nomenclature: Crl:NU(lco)-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 Mice - JAX® Mice strain - Nomenclature: CBy.Cg-Foxn1^{nu}/J

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.

BALB/c Nude Mice - Nomenclature: CAnN.Cg-Foxn1^{nu}/Crl

Origin: Developed through crosses and back-crosses between BALB/cABom-*nu* and BALB/cAnNCrj-*nu* at Charles River Japan. Pedigreed pregnant females of BALB/cAnNCrj-*nu* were received from Charles River Japan in 1985.

Characteristics: T-cell deficient. Hairless; albino.

SCID Mice - Nomenclature: CB17/ICR-Prkdc^{scid}/Crl

Origin: The *scid* autosomal recessive mutation, was detected in 1980 by MJ Bosma and his group in an inbred strain (C.BKa-*Igh^b/Icr* also known as C.B-17) of specific pathogen free mice at the Fox Chase Cancer Center, Philadelphia, PA, USA. In 1989, Charles River France obtained SCID mice under licence from FCCC.

Characteristics: B and T-cell deficient. Inbred; albino.

SHO mice - Nomenclature: Crl:SHO-Prkdc^{scid}Hr^{hr}

Origin: Developed through crosses between Crl:HA-Prkdc^{scid} and Crl:SKH1-Hr^{hr} stocks in 2007 at Charles River USA.

Characteristics: B and T-cell deficient and hairless . Outbred.



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NOD SCID Mice - JAX® Mice strain - Nomenclature: NOD.CB17-*Prkdc*^{scid}/J

Origin: Spontaneous mutation congenic on a NOD/ShiLtJ. 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) - JAX® Mice strain

Nomenclature: NOD.Cg-*Prkdc*^{scid} Il2rg^{tm1Wjl}/SzJ

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- γ (gamma c) - which is a key immune signalling molecule. Does not produce detectable serum immunoglobulin. No functional NK cell activity. Recent publications have demonstrated this strain's outstanding utility in studies of islet transplantation, haematopoietic stem cells and cancer stem cells. Inbred, albino.

SCID Beige Mouse - Nomenclature: CB17.Cg-*Prkdc*^{scid} *Lyst*^{bg}/Crl

Origin: A congenic mouse that possesses both the autosomal recessive mutations *scid* and beige. This mouse was developed by Croy *et al.*, at the University of Guelph by an intercross of C.B-17 *scid* mice to C57BL/6 *bg* mice.

Characteristics: Triple immunodeficient (B, T and NK cells). Inbred; albino.

NIH-III Mouse - Nomenclature: Crl:NIH-*Lyst*^{bg} *Foxn1*^{nu} *Btk*^{xid}

Origin: Most commonly called the NIH-III, the model was developed at the National Institute of Health, US. In addition to the T-cell deficiency, this model has two other mutations important in regulating the function of the immune system. A natural killer cell mutation, referred to as beige (*Lyst*^{bg}), renders the animal devoid of NK cells. The x-linked immune defect (*Btk*^{xid}) affects the maturation of T-independent B lymphocytes. However, the extent of the T-independent B lymphocyte and NK cell deficiencies in the NIH-III has not been fully established.

Characteristics: Triple immunodeficient (B, T and NK cells). Outbred; hairless with light to dark gray pigmented skin.

Fox Chase Outbred SCID Mouse - Nomenclature: Crl:HA(ICR)-*Prkdc*^{scid}

Origin: In 1990, the *scid* mutation was placed on the Icr:Ha(ICR) background by crossing to the C.B17/ICR-*scid* strain. Two additional backcrosses to Icr:Ha(ICR) stock were completed in 1992. The resulting Fox Chase Icr:Ha(ICR)-*scid* random bred colony is maintained by a modified version of Robertson's mating system. To CRL in 2002.

Characteristics: B and T-cell deficient. Outbred; albino.



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NMRI NUDE - 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.

Nude Rat - Nomenclature: Crl:NIH-Foxn1^{nu}

Origin: The NIH nude rat was developed in 1979-1980 through a series of matings involving 8 inbred rat strains. To CRL from the National Institutes of Health Animal Genetic Resources and caesarean rederived in 2001. This athymic model shows depleted cell populations in thymus dependant areas of peripheral lymphoid organs.

Characteristics: T cell deficient. Outbred; albino, black, black & white.



Strain	Hair	T Cells	B Cells	NK Cells	C'
CD-1 NUDE	NO	NO	YES	YES	YES
SWISS NUDE	NO	NO	YES	YES	YES
NMRI NUDE	NO	NO	YES	YES	YES
BALB/C NUDE	NO	NO	YES	YES	YES
NIH III	NO	NO	NO	NO	YES
C.B.-17 SCID	YES	NO	NO	YES	YES
ICR Outbred SCID	YES	NO	NO	YES	YES
SHO	NO	NO	NO	YES	YES
SCID-Beige	YES	NO	NO	NO	YES
NOD-SCID	YES	NO	NO	Impaired	NO
NU/NU Rat	NO	NO	YES	YES	YES
NSG	YES	NO	NO	NO	NO




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