MODELS

All Mice Are Not Created Equal
The Jackson Laboratory is the global leader in mammalian genetics modeling and education, and has been a driving force in supporting scientific breakthroughs for over 90 years. Our passion is empowering the global biomedical community in our shared quest to advance human health in therapeutic areas ranging from cancer, infectious and metabolic diseases to inflammation, neurobiology, and rare diseases.

Reach Your Next Milestone Faster

Drug development is expensive and time-consuming, with considerable failure rates. With the most innovative preclinical models and services, we enable more informed decision-making and compound evaluation to de-risk preclinical drug development and get you to your next milestone faster.

Transforming Research

Developing the most sophisticated models and applications, we are committed to providing you with the tools that propel science. Our mice and service solutions combine innovation, reliability, and commitment to customer service to offer you the most clinically-relevant mouse models and precision services. Through our industry-leading animal health and genetic stability programs, our team of scientists and mouse husbandry professionals ensures that mouse strains you receive are uniform, well-characterized, and have the highest health quality available.
Bar Harbor, Maine

Our headquarters in Bar Harbor, Maine, is home to 38 research teams who investigate the genetic basis of human disease, plus one of the world’s longest-standing programs in biomedical education and training. Each year, our production facilities provide millions of laboratory mice and a range of integrated research services to more than 19,000 biomedical and pharmaceutical researchers worldwide.

Sacramento, California

Our state-of-the-art facility in Sacramento, California, is the largest high health status mouse breeding and production facility in the United States, conveniently located near the largest biotechnology and pharmaceutical research clusters in the United States.

Farmington, Connecticut

JAX Genomic Medicine in Farmington, Connecticut, opened in 2014, focuses on medical applications of the human genome, harnessing the power of genomics to better inform preclinical drug development and clinical treatments.

*New! Ellsworth, Maine

The Charles E. Hewett Center in Ellsworth, Maine represents the culmination of nine decades of unmatched experience and leadership in caring for laboratory mice. This new facility enables wider access to vital JAX mouse resources to the worldwide biomedical research community.
BROADEST RANGE OF
MOUSE MODELS

JAX offers over 11,000 of the most common and unique strains available.

jax.org/mouse-search

Over 11,000 of the most common and unique strains available, with more than 600 new strains added each year.

Over 1,800 strains maintained live to expedite your research.

Can’t find the model you need? JAX Mouse Model Generation services can develop a unique model for your unique research.

Seamless enrollment in drug efficacy studies.
The largest collection of strains with the highest health standards.

All JAX® Mice colonies are routinely monitored to ensure their health via our JAX® Mice Animal Health Program. Over 85% of the mice we ship to researchers worldwide are from pathogen and opportunistic-free barrier rooms. No other single alternative vendor is able to deliver at this quality level.

We understand the importance of animal health in producing high-quality reproducible data. To ensure the health quality of our mice and provide you with data you can trust, we have established industry-leading rigorous animal health and monitoring programs to deliver the cleanest mice possible, including specific opportunist & pathogen free (SOPF) mice.

jax.org/animal-health
State-of-the-art facilities, surveillance, and health monitoring programs.

Building on our nearly 90-year experience, we understand the significant impact that sub-optimal animal health can have on research timelines. As a result JAX has implemented the highest operational and quality standards among mouse providers, ensuring that these standards are maintained throughout all of our distribution, client breeding and service colonies. Due to these rigorous processes, we have not experienced a significant health incident in our distribution colonies for decades. For example, the last pinworm infection at JAX dates back to the 1970’s.

Rederivation of newly imported strains.

All mice brought into The Jackson Laboratory are rederived. The imported mice are maintained in isolated barrier facilities. These quarantine activities are conducted in The Jackson Laboratory Importation Facility, which is physically separated from all production and research animals rooms. All mouse manipulations are performed in biosafety cabinets and are housed in disposable caging with negatively ventilated IVC racks.

Rederived mice are housed in microisolator cages maintained in positively ventilated IVC racks and all mice used as foster mothers in the rederivation program are from a colony with defined aerobic flora. After weaning, the mice are held until their opportunistic and pathogen- free health status is confirmed by testing foster/ surrogate mothers for all agents that we monitor.
More than 15,799 studies have been published to date using the C57BL/6J mouse.

jax.org/strain/000664

The Jackson Laboratory’s C57BL/6J (B6J) continues to be the most widely utilized inbred strain by the research and drug discovery community. The breadth and depth of scientific knowledge accumulated on this strain is unrivaled.
Diets high in fat have been attributed to many diseases, including obesity, metabolic, and inflammatory diseases. We maintain an inventory of DIO models and can place any strain on fully customized diets.

jax.org/dio

KEY BENEFITS

• Models when you need them: study-ready C57BL/6J DIO males and controls available from 7-30 weeks of age.

• Customized diets and studies can be designed for C57BL/6J or any other strain.

• Seamlessly integrate DIO models into drug studies with our In Vivo Pharmacology Services.
Aged C57BL/6J mice are used in a host of research applications, including immunology, cancer, longevity interventions, and biomarker studies. By aging the mice in our facility, we save you precious vivarium space and the headache of planning experiments years in advance.

jax.org/aged-b6

KEY BENEFITS

- Models when you need them: C57BL/6J males and females aged 25 to 78 weeks available for order immediately.
- Colonies are managed by our patented Genetic Stability Program to minimize genetic drift.
- Seamlessly integrate Aged C57BL/6J into drug studies with our In Vivo Pharmacology Services.
# Metabolic Disease Models

<table>
<thead>
<tr>
<th>Name &amp; Stock Number</th>
<th>Benefits</th>
<th>Considerations</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>B6 ob</strong> B6.Cg-Lept/J (000632)</td>
<td>- Hyperphagia</td>
<td>- An extended acclimation period helps to counteract weight loss during transit</td>
<td>Genuth et al. 1971, Dubuc 1976; Dong et al. 2006</td>
</tr>
<tr>
<td><strong>BKS db</strong> BKS.Cg-Dock +/+ Lepr/J (000642)</td>
<td>- Obesity by 4 weeks of age</td>
<td>- Hyperphagia</td>
<td>Hummel et al. 1966; Like et al. 1970; Norido et al. 1984; Wendt et al. 2003; Giacomelli et al. 1979; Werner et al. 1994</td>
</tr>
<tr>
<td><strong>B6 DIO</strong> C57BL/6J Diet-Induced Obesity (380050)</td>
<td>- Glucose intolerance with sustained hyperglycemia by 8 weeks of age</td>
<td>- Severe islet atrophy causing hypoinsulinemia and death by 10 months of age</td>
<td>Collins et al. 2004; Petro et al. 2004; Rossmeisl et al. 2003; Van Heek et al. 1997; Surwit et al. 1995</td>
</tr>
<tr>
<td><strong>B6 db</strong> B6.BKS(D)-Lepr/J (000697)</td>
<td>- Pancreatic islet atrophy</td>
<td>- Infertility (homozygous females); subfertility (homozygous males)</td>
<td>Genueth et al. 1971, Dubuc 1976; Dong et al. 2006</td>
</tr>
<tr>
<td><strong>RCS-10</strong> NONcNZO10/LtJ (004456)</td>
<td>- Hypertriglyceridemia</td>
<td>- Metabolic phenotypes seen in males only</td>
<td>Leiter and Reifsnyder 2004; Reifsnyder and Leiter 2002; Leiter et al. 2013</td>
</tr>
</tbody>
</table>

**Type 2 diabetic phenotypes include:**
- Obesity by 4 weeks of age
- Glucose intolerance by 8 weeks of age
- Transient hyperglycemia that peaks at 6-8 weeks of age
- Beta cell hypertrophy without islet atrophy
- Dysregulated adrenal medullary and pituitary gland hormone production
- Reduced thermal regulation
- Metabolic dysregulation

**Dysregulated adrenal gland hormone production**
- Medullary and pituitary gland hormone production
- Early hyperinsulinemia that peaks at 6-8 weeks of age
- Hypertension
- Selective mesenteric fat distribution
- Control mice fed a low-fat diet are available at matching ages

**Beta cell hypertrophy without islet atrophy**
- Glucose intolerance and severe hyperglycemia by 8 weeks of age
- Impaired glucose tolerance by 8 weeks of age
- Beta cell hypertrophy without islet atrophy
- Hypertension
- Selective mesenteric fat distribution
- Control mice fed a low-fat diet are available at matching ages

**Pancreatic islets transition from hypertrophy and hyperplasia to atrophy by 24 weeks**
- Metabolic phenotypes seen in males only
- Optimal phenotype development requires feeding mice a diet containing 10% fat by weight

**Type 2 diabetic phenotypes include:**
- Obesity by 4 weeks of age
- Glucose intolerance by 8 weeks of age
- Transient hyperglycemia that peaks at 6-8 weeks of age
- Insulin resistance
- Metabolic dysregulation
- Hyperphagia
- Infertility (homozygous females); subfertility (homozygous males)
- Impaired wound healing

**Hyperphagia**
- Severe islet atrophy causing hypoinsulinemia and death by 10 months of age
- Infertility (homozygous females); subfertility (homozygous males)
- Metabolic phenotypes seen in males only
- Optimal phenotype development requires feeding mice a diet containing 10% fat by weight
<table>
<thead>
<tr>
<th>TH</th>
<th>KK-Ay, KKAy</th>
<th>BTBR obese</th>
<th>BKS eNOS db</th>
<th>MS-NASH</th>
</tr>
</thead>
<tbody>
<tr>
<td>TALLYHO/JngJ (005314)</td>
<td>KK.Cg-A'J (002468)</td>
<td>BTBR.V(B6)-Lepob/WiscJ (004824)</td>
<td>BKS.Cg-Leprdb Nos3tm1Unc/RhrsJ (008340)</td>
<td>MSNASH/PcoJ (030888)</td>
</tr>
</tbody>
</table>

**Type 2 diabetic phenotypes include:**
- Moderate obesity in males and females
- Males are normoglycemic at weaning, glucose intolerant by 4 weeks, and hyperglycemic by 10–14 weeks
- Hyperinsulinemia in males by 8 weeks of age
- Pancreatic islet hypertrophy and hyperplasia observed in both sexes
- Elevated triglycerides, total cholesterol, HDL, and free fatty acids
- Males treated with high-dose Rosiglitazone have lower blood glucose and improved glucose tolerance

**Type 2 diabetic phenotypes include:**
- Moderate obesity
- Glucose intolerance and early, severe hyperglycemia
- Hyperinsulinemia and insulin resistance
- Pancreatic islet hypertrophy and hyperplasia

**Type 2 diabetic phenotypes include:**
- Obesity by 6 weeks of age
- Severe hyperglycemia by 6 weeks of age in males and 8 weeks of age in females
- Early glomerular nephropathy by 8 weeks
- Advanced diabetic nephropathy by 20–22 weeks
- Hypertriglyceridemia
- Early hyperinsulinemia that transitions to hypoinsulinemia after pancreatic islet atrophy

**Type 2 diabetic phenotypes include:**
- Obesity by 4 weeks of age
- Moderate hypertension
- Glucose intolerance and severe hyperglycemia by 8 weeks of age, comparable to BKS db
- Diabetic nephropathy
- Transient hyperinsulinemia that transitions to hypoglycemia after islet atrophy

**Type 2 diabetic phenotypes include:**
- Polygenic inbred model of obesity, diabetes, and metabolic syndrome
- Elevated blood glucose in males aged > 14 weeks fed a chow diet
- Responds to treatment with semaglutide
- A Western diet with fructose water leads to metabolic syndrome and NASH in males: enhanced weight gain, insulin resistance, high cholesterol, elevated markers of liver damage, hepatocellular ballooning, fibrosis

**Severe metabolic phenotypes are restricted to males**
- Most metabolic phenotypes are restricted to males
- Hyperphagia
- Males develop diabetic glomerular nephritis and arteriosclerosis

**Hyperphagia**
- Infertility (females); subfertility (males)

**Hyperphagia**
- Peripheral neuropathy, myocardial disease and delayed wound healing
- Islet atrophy leads to death by 10 months of age
- Infertility (females); subfertility (males)

**Variability in weight gain is observed among individuals**
- Disease is restricted to male mice only

**Kim et al. 2001, 2005, 2006; Leiter et al. 2013**
**Lan et al. 2003; Clee et al. 2005; Hudkins et al. 2010**
**Zhao et al. 2006; Hummel et al. 1966; Like et al. 1970; Norido et al. 1984; Wendt et al. 2003, Giacomelli et al. 1979, Werner et al. 1994**
**Droz et al. 2017; Peterson et al. 2017**
The most versatile immunodeficient strain enabling research advances that were not previously possible. This model engrafts a wide range of human cells and tissues, making it the strain of choice for cancer xenograft modeling and studies in immunoncology, stem cell biology, and efficacy testing.

[jax.org/strain/005557]
The triple transgenic NSG™-SGM3 mice enable superior engraftment of myeloid and CD4+ T cell lineages and primary AML samples and are useful for immuno-oncology, immunology and infectious disease studies.

jax.org/strain/013062
### IMMUNODEFICIENT MODELS

<table>
<thead>
<tr>
<th>Name &amp; Stock Number</th>
<th>NOD scid gamma (NSG™)</th>
<th>NOD Rag gamma (NRG)</th>
<th>NOD scid gamma IL3, GM-CSF, SCF (NSG-SGM3)</th>
<th>NSG™-IL15</th>
<th>NSG™ MHC Class I-null</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Name &amp; Stock Number</strong></td>
<td>NOD.Cg-Prkdc&lt;sup&gt;scid&lt;/sup&gt; I2rg&lt;sup&gt;tm4Wjl&lt;/sup&gt;/SzJ (005557)</td>
<td>NOD.Cg-Rag&lt;sup&gt;1&lt;/sup&gt;Il2rg&lt;sup&gt;tm1Mom&lt;/sup&gt; I2rg&lt;sup&gt;tm1Wjl&lt;/sup&gt;/SzJ (007799)</td>
<td>NOD.Cg-Prkdc&lt;sup&gt;scid&lt;/sup&gt; I2rg&lt;sup&gt;tm4Wjl&lt;/sup&gt;TgICMV-IL3,CSF2,KITLG1Eav/MloySzJ (013062)</td>
<td>NOD.Cg-Prkdc&lt;sup&gt;scid&lt;/sup&gt; I2rg&lt;sup&gt;tm4Wjl&lt;/sup&gt;Tg(IL15)1Sz/SzJ (030890)</td>
<td>NSG&lt;sup&gt;™&lt;/sup&gt;-B2m (010636) NSG-(K&lt;sup&gt;a&lt;/sup&gt;D&lt;sup&gt;1&lt;/sup&gt;)null (023848)</td>
</tr>
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</table>

| Mature B cells | Absent | Absent | Absent | Absent | Absent |
| Mature T cells | Absent | Absent | Absent | Absent | Absent |
| Dendritic Cells | Defective | Defective | Defective | Defective | Defective |
| Macrophages | Defective | Defective | Defective | Defective | Defective |
| Natural killer cells | Absent | Absent | Absent | Absent | Absent |
| Complement | Absent | Absent | Absent | Absent | Absent |
| Leukiness | Very low | Absent | Absent | Negligible | Negligible |
| Irradiation tolerance | Low | High | Low | Low | Low |
| Lymphoma incidence | Low | Low | Low | Low | Low |

**Benefits**
- Engrafts the widest range of solid and hematological cancers, including ALL and AML
- Most sensitive host for cancer stem cells when compared to NOD scid or nude mice
- Longer lifespan than NOD scid; supports long-term engraftment studies and capabilities; >89 weeks median survival
- No thymic lymphomas—can be used for long and short-term experiments
- Sensitive to irradiation
- Engrafts human PBMC without irradiation similar to NSG™
- Long-term multilineage hematopoietic stem cell repopulation similar to NSG™ mice
- Engrafts a wide range of solid and hematological cancers
- Compromised human stem cell regeneration
- Suppression of human erythropoiesis
- Reduction of human B-lymphopoiesis
- Increased CD4+ FoxP3+ regulatory T cell population
- Enhanced human myelopoiesis and terminal differentiation,
- Increased efficiency of engrafting human acute myeloid leukemia (AML)
- Increased abundance and function of human NK cells following CD34-humanization
- Resistant to xeno-GVHD (10636)
- Attenuated xeno-GVHD development post- hPBMC transplantation (023848)
- High hCD45+ cell engraftment (023848)
- Useful for studying mechanisms for xeno-GVHD

**Considerations**
- Reduced survival post hCD34+ cell transplantation compared to NSG mice (023848)

**References**
- Ishikawa et al. 2005 (PMID: 15920010)
- Shultz et al. 2005 (PMID: 15879151)
- Pearson et al. 2008 (PMID: 18785974)
- Brehm et al. 2010 (PMID: 20096637)
- Nicolini et al. 2004 (PMID: 14628073)
- Wunderlich et al. 2010 (PMID: 20686503)
- Maykel et al. 2014 (PMID: 24798995)
- Billerbeck et al. 2015 (PMID: 21252091)
- Brehm et al. 2018 (In: Immunology 2018 Meeting Abstracts)
- Covassin et al. 2013 (PMID: 23869841)
### Class 1/Class 2 Knockout MHC I/II KO

<table>
<thead>
<tr>
<th>NOD.Cg-Prkdcsid</th>
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<tbody>
<tr>
<td>H2-Ab1em1Mew H2-K1em1Bpa</td>
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<tr>
<td>H2-D1tm1Bpe II2rgtm1Mo/szJ</td>
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<table>
<thead>
<tr>
<th>NOD scid</th>
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<tbody>
<tr>
<td>NOD.CB17-Prkdcsid/J</td>
</tr>
<tr>
<td>CBySrn.CB17-Prkdcsid/J</td>
</tr>
<tr>
<td>B6.129S7-Rag1™mm1Mom/J</td>
</tr>
<tr>
<td>J:NU (007850)</td>
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<td>NU:J (002019)</td>
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<tr>
<td>Negligible</td>
<td>Very low</td>
<td>Very low</td>
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<td>Low</td>
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<td>Low</td>
<td>High</td>
<td>High</td>
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<tr>
<td>Low</td>
<td>High (thymic lymphoma)</td>
<td>High (thymic lymphoma)</td>
<td>Low</td>
<td>Low</td>
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</table>

- Most resistant to xeno-GVHD of all NSG variants
- Extended human IgG half-life compared to B2m knockouts
- Most radiation sensitive
- Higher take-rates for slow-growing cancer cell lines than BALB scid or nude xenograft models
- Xenotransplantation of some solid human tumors
- Adaptive transfer from strains on NOD background enables study of cell function & track cell movement
- About 36 weeks median survival

- Allows allogeneic and xenogeneic cancer cell lines & tissues
- Engrafts hematopoietic cancer cell lines, some primary cells
- Improvements in engraftment efficiency over nude models for some cancer cell lines
- Radiation resistant, providing an alternative to scid mutants
- Adoptive transfer from strains on B6 background permits to study cell function and track cell movement

- Engraftment of human & mouse tumor cell lines
- Easy assessment of subcutaneous tumor growth due to lack of fur
- Resistance to xeno-GVHD associated with reduced CD45+ cell expansion in vivo
- Radiation sensitive
- Develops thymic lymphomas by 8-9 months—best used in short-term experiments
- Sensitive to irradiation
- Innate immunity intact
- NK cell activity limits engraftment
- Sensitive to irradiation
- Innate immunity intact
- Poor host for primary cell transplantation
- Innate immunity intact
- Little engraftment of hematopoietic cancer cells
- Not suitable for primary cell transplantation

**References**

- Brehm et al. 2018 (PMID: 30383447)
- Shultz et al. 1995 (PMID: 7995938)
- Nonyama et al. 1993 (PMID: 8473734)
- Mombaerts et al. 1992 (PMID: 1547488)
Strains with HLA-Restricted hu-CD8+ T Cells

NSG HLA Class I transgenic strains

**NSG HLA-A2.1**
NOD.Cg-Prkdc<sup>scid</sup>Il2rg<sup>tm1Wjl</sup>Tg(HLA-A2.1)1Enge/SzJ 009617

This model enables the study of immune response to human antigens overexpressed in cancer cells, representing a unique preclinical model for cancer vaccine development and efficacy testing of immune checkpoint modulators.

[jax.org/strain/009617](jax.org/strain/009617)

**NSG-HLA-A2/HHD**
NOD.Cg-Prkdc<sup>scid</sup>Il2rg<sup>tm1Wjl</sup>Tg(HLA-A/H2-D/B2M)1Dvs/SzJ 014570

NSG-HLA-A2/HHD mutant mice are immunodeficient and express human HLA class 1 heavy and light chains. This strain may be useful as a human hematopoietic engraftment host that supports the maturation of human T cells with transplantation of human hematopoietic stem cells.

[jax.org/strain/014570](jax.org/strain/014570)

Strains with Increased hu-CD4+ T Cell and Antibody Responses

NSG HLA Class II transgenic strain

**NSG-Ab<sup>o</sup> DR4**
NOD.Cg-Prkdc<sup>scid</sup>Il2rg<sup>tm1Wjl</sup>H2-Ab<sup>1tm1Gru</sup>Tg(HLA-DRB1)31Dmz/SzJ 017637

NSG-HLA-A2/HHD may be used as a preclinical model for cancer vaccine development and efficacy of immune checkpoint modulators. The expression of the transgene establishes a humanized immune microenvironment allowing functional maturation of human HSC. This strain is useful as a human hematopoietic engraftment host that supports the maturation of human T cells.

[jax.org/strain/017637](jax.org/strain/017637)

**DRAG**
NOD.Cg-Rag<sup>1tm1Mom</sup>Il2rg<sup>tm1Wjl</sup>Tg(HLA-DRA,HLA-DRB1*0401)39-2Kito/ScasJ 017914

This model exhibits enhanced HLA-DR-matched hematopoietic stem cell engraftment and subsequent human T cell and B cell development. This strain may be useful as an in vivo model for studying human T cell/B cell development and function, vaccine testing, and generation of "fully human" IgM, IgG, IgA, or IgE monoclonal antibodies for prophylactic and/or therapeutic use in autoimmune diseases and infectious diseases.

[jax.org/strain/017914](jax.org/strain/017914)
Strains Expressing Human Growth Factors That Support Human Hematopoiesis

**NSG-Tg(hu-mSCF) NOD.Cg-Prkdc<sup>scid</sup> Il2rg<sup>tm1WjI</sup> Tg(PGK1-KITLG*220)441Daw/SzJ 017830**
This host will allow myeloid engraftment (especially mast cells) to study human mast cell development and allergic responses to immuno-oncology therapeutic approaches with the practical advantage of not requiring pre-conditioning irradiation before engraftment (Brehm et al. 2012 [PMID: 22246028]; Takagi et al. 2012 [PMID: 22279057]).
[jax.org/strain/017830](https://jax.org/strain/017830)

Strains Useful in Researching Human Versus Mouse Stroma

**NSG hprt-null NOD.Cg-Prkdc<sup>scid</sup> Il2rg<sup>tm1WjI</sup> Hprt<sup>-m3J</sup>/EshJ 012480**
This strain enables you to speed the process of isolating and establishing patient-derived cancer cell lines for drug screening programs. It greatly facilitates the isolation of human cancer cells from mouse stroma in vitro, and enables establishment of new patient-derived cell lines (Kamiyama et al. 2013 [PMID: 23340293]).
[jax.org/strain/012480](https://jax.org/strain/012480)

**NSG-EGFP NOD.Cg-Prkdc<sup>scid</sup> Il2rg<sup>tm1WjI</sup> Tg(CAG-EGFP)1Osb/SzJ 021937**
This strain retains the same immunodeficiency phenotypes as NSG™ but also expresses enhanced green fluorescent protein (EGFP) in nearly all tissues. EGFP allows tracking of mouse-derived stroma or residual innate immune cells in engrafted tumors and tissues. (Maykel et al. PMID: 24798995)
[jax.org/strain/021937](https://jax.org/strain/021937)

Additional Strains

**NBSGW NOD.Cg-Kit<sup>W-41J</sup> Tyr<sup>*</sup> Prkdc<sup>scid</sup> Il2rg<sup>tm1WjI</sup>/ThomJ 026622**
This model supports engraftment of human hematopoietic stem cells without irradiation, exhibits longer lifespan that other immunodeficient Kit mutants, and has increased human erythroid cells (bone marrow). This strain is suitable for humanization experiments involving CD34+ stem cells.
[jax.org/strain/026622](https://jax.org/strain/026622)

**NSG-PiZ NOD.Cg-Prkdc<sup>scid</sup> Il2rg<sup>tm1WjI</sup> Tg(SERPINA1*E342K)#Slcw/SzJ 028842**
This strain expresses the mutant human SERPINA1 on the immunodeficient NSG™ background, which provides a stable engraftment environment for primary human hepatocytes after partial heptectomy. This strain may be useful in studies relating to humanized liver xenografts.
[jax.org/strain/028842](https://jax.org/strain/028842)
Humanized NSG™ mice represent an innovative and cost-effective platform to simulate trials, evaluate multiple drugs alone or in combination, and produce predictive data.

**hu-PBMC**

Created with adult peripheral blood mononuclear cells, the hu-PBMC models feature quick engraftment and enable short-term studies requiring mature human T cells.

**hu-CD34**

Produced by engraftment of hu-HSCs, these models yield robust multilineage immune systems with T cell maturation and function for long-term studies.
**MODEL COMPARISON GUIDE**

<table>
<thead>
<tr>
<th>MODEL</th>
<th>hu-PBMC-NSG™</th>
<th>hu-CD34-NSG™</th>
<th>hu-CD34-SGM3</th>
<th>hu-CD34-NSG™-IL-15</th>
</tr>
</thead>
<tbody>
<tr>
<td>STRAIN</td>
<td>NSG™ (005557)</td>
<td>NSG™ (005557)</td>
<td>NSG™-SGM3 (013062)</td>
<td>NSG™-IL-15 (003089)</td>
</tr>
<tr>
<td>HUMAN CYTOKINE EXPRESSION</td>
<td>N/A</td>
<td>N/A</td>
<td>KITL (SCF), IL3, GM-CSF</td>
<td>IL-15</td>
</tr>
</tbody>
</table>
| BENEFITS   | • Enables short-term studies requiring human T cells.  
            • Strong effector and memory T cell function.  
            • T cell-driven GvHD.  
            • A functional immune system.  
            • T cell-dependent inflammatory responses.  
            • No donor cell immune reactivity towards host.  
            In addition to benefits of hu-CD34-NSG™:  
            • Faster immune cell repopulation*.  
            • Higher myeloid cell engraftment*.  
            • Faster lymphoid cell engraftment*.  
            • Higher T cell and dendritic cell populations*.  
            • Enhanced AML engraftment*.  
            • In addition to the benefits of hu-CD34-NSG™:  
            • Faster immune cell repopulation*  
            • Higher natural killer cell engraftment*  
            • Natural killer cells at human physiological levels |
| LIFESPAN   | Short-term <3 months | Long-term >12 months | Long-term >12 months | Long term >12 months |
| ENGRAFTMENT STABILITY | Stable through the lifespan of the mouse. | Stable through the lifespan of the mouse. | Stable through the lifespan of the mouse. | Stable through the lifespan of the mouse |
| IMMUNE DEVELOPMENT | T cells (HLA-restricted CD4 and CD8), Natural Killer cells.  
                       B, T (MHC-restricted CD4 and CD8), Monocytes, Macrophages, Dendritic cells.  
                       T cells (MHC-restricted CD4 and CD8), Monocytes, Macrophages, Dendritic cells.  
                       T cells (MHC-restricted CD4 and CD8), Monocytes, Macrophages, Dendritic Cells, Natural Killer Cells. |
| AVAILABILITY | Readily Available | Readily Available | Readily Available | Readily Available |

*When compared to hu-CD34-NSG™*

Study ready hu-NSG™ and hu-NSG™-SGM3 cohorts are available immediately for shipments worldwide.

Learn more by visiting [jax.org/humanized-mice](http://jax.org/humanized-mice)
Onco-Hu™ models are a robust immuno-oncology platform for efficacy testing of novel immunotherapies targeting T cells and myeloid cells to help destroy cancers *in vivo*.

The Onco-Hu™ platform is based on NSG™ and NSG™-SGM3 mice, dually engrafted with human CD34+ hematopoietic stem cells (HSC) and clinically relevant PDX Live™ low passage tumors.

**Advantages**

Onco-Hu™ better recapitulates human tumor and immune cell interactions *in vivo* compared to existing models, allowing improved physiological modeling of pathways important in therapeutic intervention. In addition to robust engraftment of low passage PDX tumors, the Onco-Hu™ model stably expresses diverse human immune cells, including:

- Hematopoietic stem cells *(CD4+)*
- Myeloid progenitor cells
- CD33+ myeloid cells
- Dendritic cells
- T-helper cells
- T cytotoxic cells
- T reg cells
- B cells

Onco-Hu™ models that express PD-L1 respond to anti-PD-1 check-point inhibitors, inducing tumor infiltration of cytotoxic T cells and significant reduction of tumor growth rates compared to vehicle-treated control mice (Li-Chin Yao, et al. 2015).
Revolutionizing Pre-Clinical Research

WE RUN ACCELERATED EFFICACY STUDIES FOR YOU

Our In Vivo Pharmacology Services offers optimized immuno-oncology efficacy studies to test anti-tumor responses using highly characterized Onco-Hu™ models expressing high PD-L1 levels. These validated platforms support robust tumor growth and respond to check-point inhibitors, such as anti-PD-1 receptor antibody pembrolizumab exhibiting tumor infiltrating cytotoxic T cells and reduction of tumor growth rate.

Onco-Hu™ models engrafted with PDX Live™ clinically relevant breast or lung tumors allow the evaluation of the efficacy of immunomodulators –alone or in combination therapies– to treat cancer.

jax.org/onco-hu
A UNIQUE MODEL FOR
YOUR UNIQUE RESEARCH

Utilizing the right in vivo model can be the difference between achieving your milestone and starting your project over. Leverage JAX scientific expertise to generate the best models on the most appropriate background for your research. Only JAX maintains a comprehensive collection of mouse models and understands the scientific complexities of each strain.

Translationally-Relevant Models

Developing a model you need on a genetically stable background, at a high health status will eliminate the need to quarantine, rederive, or backcross your mice. JAX Project Scientist collectively have decades of experience generating new models on difficult backgrounds, including NOD and NSG™ mice.
Study Reproducibility

As a result of the JAX comprehensive design process and rigorous validation approach — which includes a search for random integrants and using our patented Genetic Stability Program — we have a 97% success rate delivering genetically-validated, germline component models. Our program significantly reduces the risk of genetic drift, ensuring that the model used in your studies is reproducible.

Engineering Value

JAX is the premier institution using mouse models to study biology, genetics, and disease. As scientists, we have the breadth of experience, the selection of high health status mouse models, knowledge of strain biology and JAX GSP, and the know-how to add value to your research plan.

jax.org/model-generation
**Mouse Phenome Database**

Harness the decades of published data on JAX® Mice. The Mouse Phenome Database (MPD), a free, continually expanding, and collaborative bioinformatics resource from JAX, is focused on collecting standardized experimental data from across many strain backgrounds and research areas.

*phenome.jax.org*

**ACCESS JAX® MICE CHARACTERIZATION DATA**

With MPD, don’t re-characterize models – generate new discoveries!

MPD holds data from more than 447 different JAX strains, including 46 of the most commonly used inbred mice.
MPD saves scientists time and resources.

MPD includes downloadable baseline phenotypic data sets for parameters such as body weight, blood chemistry, disease susceptibility, reproductive rates and gene expression as well as studies on the effects of different drugs, diet, disease and aging on mice. Detailed protocols on data collection, project descriptions, and related publications are also available to facilitate experimental reproducibility.

With MPD, you can:

- Identify the best genetic background for creating new models
- Compare your data to previous findings
- Access whole-genome sequencing data for 27 JAX strains
- Understand expected baseline strain phenotypes
- Perform strain-to-strain comparisons
- Review SNPs and indels for 29 JAX strains
Reproducibility is the foundation of new scientific discovery; the challenge is that living organisms change. Random mutations in mouse colonies lead to genetic variation and drift of the research tools you rely on to be genetically identical. Mutations become fixed over time and change the phenotype of mouse strains vital to research programs. If left unattended, it is just a matter of time before mutations affect an experiment in unexpected ways (Stevens et al., 2007).
We have overcome the cumulative genetic drift challenge by developing and implementing our innovative Genetic Stability Program (GSP). This program effectively stops cumulative genetic drift in our most popular inbred strains by regularly refreshing foundation stocks with genetically-defined stock of frozen embryos. The GSP produces premium grade research mice with stable genomes that yield the highest level of experimental reproducibility available.

GSP-PROTECTED

- C57BL/6J (000664)
- C57BL/6NJ (005304)
- BALB/cByJ (001026)
- 129S1/SvImJ (002448)
- C3H/HeJ (000659)
- DBA/1J (000670)
- DBA/2J (000671)
- FVB/NJ (001800)
- NOD/ShiLtJ (001976)
- B6.129P2-Apoε^tmUnc/J (002052)
- CBA/J (000656)
- NOD.CB17-Prkdc^scid/J (001303)

Keep your research reproducible for generations to come. GSP-protected JAX® Mice you receive today will be the same as those you and your colleagues receive 25 years from now.

jax.org/genetic-stability
MOUSE SOURCING YOU CAN RELY ON

We’ve never canceled a shipment based on weather.

Ensuring a Stable Environment

Mice are transported by drivers thoroughly trained in animal welfare and mouse handling procedures.

- Mice are maintained under strict closely monitored environmentally-controlled conditions.
- Long-distance transports are constantly monitored for speed and acceleration to ensure that the mice have the smoothest ride possible.
- Mice arrive with minimal environmental disturbance and the least possible stress.
- Animal health barriers are maintained unbroken from packing to delivery.
WE DELIVER ANYWHERE YOU ARE.

Innovation is challenging—sourcing mice needn’t be. How reliable are we? 99% of our deliveries arrive on time.