MODELS AND SERVICES DESIGNED TO TAKE YOUR STUDY FURTHER

Oncology

TA CONIC

MODELS AND SERVICES DESIGNED TO TAKE YOUR STUDY FURTHER
Oncology

Evaluating the response of translational rodent models to new cancer therapies is the key to developing innovative treatment options.

The super immunodeficient CIEA NOG mouse® is the ideal model for engraftment of human cells, and therefore the model of choice for combined immune system and tumor engraftment immuno-oncology experiments.

Taconic Biosciences offers a comprehensive portfolio of translational rodent models to accelerate and enhance your research in the field of oncology. Mouse models available exclusively from Taconic include human immune system engrafted mice for tumor grafting and therapeutics testing; spontaneous tumor models for breast and colon cancer research; and a wide variety of immunodeficient mice, including the super immunodeficient CIEA NOG mouse® and the HRN™ nude mouse. Taconic also provides integrated model generation and breeding services to accelerate drug discovery and development timelines.
TACONIC OFFERS THREE HUMANIZED IMMUNE SYSTEM MODELS

CIEA NOG mouse®

Immunodeficient mice carrying a reconstituted human immune system.

The super immunodeficient CIEA NOG mouse® is the ideal model for engraftment of human cells, and therefore the model of choice for combined immune system and tumor engraftment immunoncology experiments.

When reconstituted with various human tissue sources, NOG mice are indispensable for basic research probing the human immune system. Engrafted NOG mice enable efficacy testing of immunotherapies as well as the unprecedented ability to study tumor specific modulation of the immune system. Taconic offers study-ready cohorts of hematopoietic stem cell-engrafted NOG mice.

In addition to the models, Taconic offers access to scientific expertise on use of the CIEA NOG mouse® for engraftment and reconstitution with human tissues.

HOW CAN IMMUNE SYSTEM ENGRAFTED NOG MICE BE USED?

Immune system engrafted mouse models are excellent tools to evaluate the effect of human immune cells in preclinical oncology:

• Assessment of therapeutic immunomodulatory activities
• Evaluation of antitumor activity related to antibody dependent cell cytotoxicity (ADCC)
• Analysis of innate and adaptive immunity
• Cytokine readouts

These models are also excellent tools for other research application, such as:

• GvHD (Graft versus Host Disease)
• T cell activation model
• B cell depletion studies
• Autoimmune disease
• Allergy
• Inflammation
• Infectious disease (HIV)
• Vaccine development
• Transplantation
• Study of hematopoiesis
**huPBMC-NOG**
NOG MICE ENGRAFTED WITH HUMAN PBMCs (PERIPHERAL BLOOD MONONUCLEAR CELLS)

- Model for investigation of adult/mature cell populations.
- Use is limited to short term studies.
- GvHD response can be used as a screening system for T cell modulating drugs.
- Available with normal or patient-derived PBMCs.

**huNOG**
NOG MICE ENGRAFTED WITH HUMAN CD34+ HEMATOPOIETIC STEM CELLS (HSCs)

- Stable engraftment of multiple cell lineages by 12-16 weeks post-injection.
- Only mice with ≥25% hCD45+ in peripheral blood are delivered.
- Long-term studies possible.
- huNOG ARE AVAILABLE OFF-THE-SHELF! PLACE YOUR ORDER NOW FOR IMMEDIATE DELIVERY.

**BLT-NOG**
NOG MICE INJECTED WITH CD34+ HEMATOPOIETIC STEM CELLS (HSCs) AND SURGICALLY ENGRAFTED WITH DONOR MATCHED THYMUS AND LIVER SECTIONS

- Stable engraftment of multiple cell lineages, with enhanced T and B cell function.
- Particularly suited to vaccine studies, antibody generation studies, and the development of helper T cell and B cell responses.

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**LICENSING:** NO MTA OR LICENSE FEE IS REQUIRED FOR ANY OF THE MODELS FEATURED ON THIS PAGE.
ONCOLOGY

IMMUNODEFICIENT MODELS

NUDES

THE AUTOSOMAL RECESSIVE NUDE GENE IN HOMOZYGOUS (NU/NU) MICE CAUSES THE LACK OF FUR AND AN ABNORMAL THYMUS. HETEROZYGOUS (NU/+ ) ANIMALS CARRY THE RECESSIVE NUDE GENE ON ONE CHROMOSOME ONLY, AND THEREFORE HAVE A NORMAL THYMUS-TRIGGERED IMMUNE SYSTEM

B6 nude

T CELL DEFICIENT MOUSE

- Foxn1nu mutation backcrossed to the C57BL/6NTac inbred strain for ten generations.

MODEL NUMBER B6NU

BALB/c nude

T CELL DEFICIENT MOUSE

- Foxn1nu mutation backcrossed to the BALB/cAnN inbred strain for nine generations.
- Available at two health designations: Defined Flora from gnotobiotic isolators and Restricted Flora™ from Isolated Barrier Units™.

MODEL NUMBER BALBNU

NCr nude

T CELL DEFICIENT MOUSE

- Outbred background originated from an accidental cross between the BALB/c.
- Inbred nude and NIH(S) outbred nude mice.
- The standard athymic model for National Cancer Institute (NCI) studies as well as many pharmaceutical and institutional oncology screening programs.

MODEL NUMBER NCRNU

TO ORDER

US: 1-888-822-6642 | EU: +45 70 23 04 05 | INFO@TACONIC.COM
NIH nude
T CELL DEFICIENT RAT

- In this outbred immunodeficient model the vibrissae are present in the homozygous nude rat, but they are bent, with some short hairs on the head and occasionally on the rest of the body.
- Good xenograft host for many cell lines.

MODEL NUMBER NIH-RNU

NMRI nude
T CELL DEFICIENT MOUSE

- Foxn1nu mutation backcrossed to the NMRI outbred stock.
- Widely-used as host for transplanted human tumors and for therapeutic studies on human tumors.
- The NMRI nude has a relatively low take-rate for human breast tumors compared to other nude or immunodeficient mice.

MODEL NUMBER NMRINU

HRN™ nude
T CELL DEFICIENT MOUSE

- The combination of the nude mutation and the HRN™ mutations permits xenograft studies in a mouse without liver P450 metabolism.
- Useful when studying highly cleared chemotherapeutics, allowing efficacy testing without the need for multiple dosing or the use of constant infusion pumps.
- Use to get a quick readout on the efficacy of your anticancer lead compounds without having to first work through PK issues.
- A combination between Taconic’s leading outbred nude, the NCr nude, and the HRN™ transgenic mouse.

MODEL NUMBER 9066
SCIDs
SEVERE COMBINED IMMUNODEFIciency

Mice homozygous for the Prkdc^{scid} mutation lack both T and B cells due to a defect in V(D)J recombination. This feature makes these models ideal for accepting foreign tissue transplants, including human tumors. These models are used for testing new cancer treatments, and as hosts for human immune system tissues (i.e., SCID-HU).

C.B-17 scid
T & B CELL DEFICIENT MOUSE

• The original congenic background strain on which Dr. Mel Bosma discovered the spontaneous scid mutation.
• Available at two health designations:

MODEL NUMBER CB17SC

ICR scid
T & B CELL DEFICIENT MOUSE

• Equivalent to the C.B-17 scid in severity of immunodeficiency, but this outbred background exhibits a significantly reduced incidence of spontaneous Ig production (leakiness).

MODEL NUMBER ICRSC

NOD scid
T & B CELL DEFICIENT MOUSE

• The scid mutation has been transferred onto a diabetes-susceptible Non-Obese Diabetic (NOD) making it a great model for diabetes and obesity research in the context of Insulin-Dependent Diabetes Mellitus (Type I diabetes).
• The multiple defects in immunity unique to this model provide a very good system for reconstitution with human hematopoietic cells, resulting in excellent models for HIV-1 research and gene therapy.
• Useful model for investigating increased tumor incidence, particularly lymphomas and thymic tumors.
• Does not develop spontaneous diabetes.

MODEL NUMBER NODSC
Rag2 MODELS

MICE HOMOZYGOUS FOR THE RAG2 NULL MUTATION EXHIBIT TOTAL INABILITY TO INITIATE V(D)J REARRANGEMENT AND FAIL TO GENERATE MATURE T OR B LYMPHOCYTES. NEVERTHELESS, THE RAG2 MOUSE HAS APPARENTLY NORMAL HEMATOPOIESIS. RAG2 KNOCKOUTS ARE USEFUL FOR VACCINE DEVELOPMENT, TRANSPLANTATION OR XENOGRAFT STUDIES, AND HEMATOPOIESIS RESEARCH. THE RAG2 MOUSE IS USEFUL IN EVALUATING THE FUNCTION OF SPECIFIC GENES AS THEY RELATE TO BONE MARROW TRANS-COMPLEMENTATION ASSAYS.

Rag2 (129S6)
T & B CELL DEFICIENT MOUSE

• The 129S6 strain was the original strain in which the Rag2 targeted mutation was created.

MODEL NUMBER RAG2

Rag2 (BALB/c)
T & B CELL DEFICIENT MOUSE

• Backcrossed twelve generations (N12) to the BALB/cAnNTac inbred strain.

MODEL NUMBER 601

Rag2 (B6.SJL)
T & B CELL DEFICIENT MOUSE

• Similar to C57BL/6 with the H2b haplotype but carries the Ptprcp and Pep3p genes from the SJL strain (CD45.1).

MODEL NUMBER 461

Rag2 (C57BL/6)
T & B CELL DEFICIENT MOUSE

• Backcrossed twelve generations (N12) to the C57BL/6NTac inbred strain.

MODEL NUMBER RAGN12
Scid-beige

**T, B & NK Cell Deficient Mouse**

- The mutations were backcrossed seven generations to the congenic C.B-17 background.
- This double mutant model carries the scid mutation which causes a lack of both T and B lymphocytes due to a defect in V(D)J recombination.
- It also carries the beige mutation which results in cytotoxic T cell and macrophage defects as well as selective impairment of NK cell functions.

**MODEL NUMBER CBSCBG**

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Rag2/Il2rg Double Knockout Mouse

**T, B & NK Cell Deficient Mouse**

- Useful for transplanting allogeneic or xenogeneic stem cells, which are often rejected by NK cells.
- May be used in combination with parent Rag2 knockout model for defining the role of NK cells in host resistance to tumors and infectious agents.
- May not be the best choice for experiments involving humanization of the immune system, since human hematopoietic stem cells do not engraft and differentiate well in strains on B6 or B6-related backgrounds.
- The Il2rg gene is located on the X chromosome, so male knockouts are hemizygous for the Il2rg mutant allele.

**MODEL NUMBER 4111**
CIEA NOG mouse®

T, B & NK CELL DEFICIENT

- The CIEA NOG mouse® is a super immune deficient mouse with unparalleled potential to engraft human cells and tissues.
- This severely immunocompromised mouse carries the scid mutation and a targeted mutation of the Il2rg gene on the NOD/ShiJic genetic background.
- The functional knockout of the IL2 receptor common gamma chain (IL2rg) results in reduction of residual innate immunity of the NOD/ShiJic background and superior engraftment of human cells and tissues compared to any other immune deficient model.
- Lack of mature T, B and NK cells, reduced complement activity, dysfunctional macrophages and dendritic cells, and deficiencies in immune signaling, including impaired cytokine production.
- Excellent choice for xenograft studies using cell lines with poor take rates in nudes or scids, or for engraftment of patient-derived tumors.
- The best choice for human immune system engraftment mice, with successful engraftment of various tissues such as PBMCs or umbilical cord blood stem cells.
- Test therapeutic antibodies and immune-modulating treatments by combining immune system engraftment of the immune system with xenograft of tumor cell lines or patient-derived tumors.
- Displays a very low incidence of lymphoma, unlike NOD scid model.
- The Il2rg gene is X-linked, so male knockouts are hemizygous for the Il2rg mutant allele.

MODEL NUMBER NOG

CIEA BRG mouse

T, B & NK CELL DEFICIENT

- Higher radiation tolerance due to Rag2 mutation compared to scid models (similar to wild type mice).
- Model is completely congenic on BALB/c background, the preferred strain background for many immunology studies.
- Applications in studies on immune system engraftment, infectious diseases and autoimmune diseases as well as cancer xenografts.

MODEL NUMBER 11503

NOMENCLATURE
C.Cg-Rag2tm1Fwa Il2rgtm1Sug/JicTac

Sponsored by the Central Institute for Experimental Animals and In-Vivo Science International.
Invasive Lobular Breast Cancer Model

**BREAST CANCER MODEL**

- Tissue-specific conditional knockout of Cdh1 (E-cadherin) and Trp53 in mice induces metastatic mammary carcinomas that resemble human invasive lobular carcinoma (ILC), the second most common type of primary breast cancer.
- From the literature, it is estimated that females develop multiple skin and mammary tumors with a median latency of 214 days.
- This mouse model provides a valuable tool to gain insights into the role of E-cadherin loss of function in mammary tumor initiation, progression, and metastasis.
- Can be used to supply tumor tissues for allografts.
- 20-30% of mice will develop non-mammary epithelial tumors.

**MODEL NUMBER** 11509

---

Brca1-Associated Breast Cancer Model

**BREAST CANCER MODEL**

- Conditional mouse mutant with somatic deletion of Brca1 and Trp53 in several epithelial tissues including mammary epithelium. Female mice of this strain show a high incidence of mammary tumors that mimic many aspects of human BRCA1-mutated basal-like breast cancer.
- Contains conditional disruption of the Brca1 gene. Germline mutations of this gene are responsible for 40% to 50% of hereditary breast cancers.
- Contains conditional disruption of the Trp53 tumor suppressor gene, the most commonly mutated gene in human cancers.
- From the literature, it is estimated that 80% of females develop multiple mammary and skin epithelial tumors with onset between 140 and 280 days.
- This model may be helpful in predicting responses of human BRCA1-deficient tumors to therapies.
- Can be used to supply tumor tissues for allografts.
- 20-30% of mice will develop non-mammary epithelial tumors.

**MODEL NUMBER** 11510

---

**NOMENCLATURE**

**Invasive Lobular Breast Cancer Model**
FVB.Cg-Cdh1<sup>tm1Jjon</sup> Trp53<sup>tm1Brn</sup> Tg(KRT14-cre)<sup>8Brn/A</sup>

---

**Brca1-Associated Breast Cancer Model**
STOCK Trp53<sup>tm1Brn</sup> Brca1<sup>tm1Brn</sup> Tg(KRT14-cre)<sup>8Brn</sup>
Floxed Ink4a/Arf Mouse
CONDITIONAL TUMOR SUPPRESSOR ALLELE

• Contains a targeted mutation of Cdkn2a (Ink4a/Arf) which introduced LoxP sites upstream of exon 2 and downstream of exon 3.
• Cross with the tissue-specific cre of your choice to develop a tumor model.

Model Number 11511

Floxed p53 Mouse
CONDITIONAL TUMOR SUPPRESSOR ALLELE

• Contains a targeted mutation of Trp53 which introduced LoxP sites flanking exons 2 through 10.
• Cross with the tissue-specific cre of your choice to generate a conditional disruption of the Trp53 tumor suppressor gene, the most commonly mutated gene in human cancers.

Model Number 11512

The cell cycle inhibitory protein Cdkn2a is frequently disrupted in various types of human cancer, and germline mutations of this locus can confer susceptibility to melanoma and other tumors.

After deletion of the gene via crossing to a tissue-specific cre line, mice can develop tumors, giving rise to various sarcomas, carcinomas, lymphomas and metastatic melanoma.

After deletion of the gene via crossing to a tissue-specific cre line, the incidence and the spectrum of tumors observed in homozygous or heterozygous mutant animals were comparable to those found in constitutive knockouts.
SPONTANEOUS TUMOR MODELS

Pirc
COLON CANCER MODEL

• Excellent model for study of human familial colon cancer.
• ENU-induced point mutation results in a truncating mutation in the Apc gene at a site corresponding to the human mutation hotspot region of the gene.
• Heterozygotes develop multiple tumors in the small intestine and colon by 2-4 months of age.
• Pirc tumors closely resemble those in humans in terms of histopathology, morphology, and distribution between intestine and colon.
• Longer lifespan compared to related mouse models (12-15 months).
• Tumors may be visualized by CT, endoscopy or dissection.
• Available for immediate cryorecovery.

MODEL NUMBER PIRC

Stat 1
MAMMARY TUMOR MODEL

• Contains a homozygous disruption of the Stat1 gene and complete lack of functional STAT1 proteins.
• The JAK-STAT signaling pathway has been implicated in mediating biologic responses induced by many cytokines.
• Deficient immune cell response to alpha and gamma interferons.
• Accelerated and amplified development of chemically-induced and spontaneous tumors.
• Useful in unraveling the role of a variety of cytokines in immune responses, the role of STAT1 protein in mediating interferon-dependent responses, and the roles of tumor cells and immune cells in mediating tumor cell destruction.

MODEL NUMBER 2045
TSG-p53®
TUMOR SUPPRESSOR KNOCK OUT

• Contains a disruption of the Trp53 tumor suppressor gene, the most commonly mutated gene in human cancers.
• Useful for studying Trp53 gene function or screening potential cancer intervention therapies.
• Homozygous TSG-p53® mice are totally deficient in p53 protein and prone to the development of spontaneous tumors, primarily lymphomas and sarcomas.
• Heterozygous TSG-p53® mice carry one normal p53 allele and have a much lower rate of spontaneous tumor development.

MODEL NUMBER P53N12
# COMPARISON OF ONCOLOGY MOUSE AND RAT MODELS

<table>
<thead>
<tr>
<th>MODEL NUMBER</th>
<th>MODEL NAME</th>
<th>COAT COLOR</th>
<th>T,B &amp; NK CELL DEFICIENCES</th>
<th>OTHER IMMUNODEFICIENCIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>BALBNU</td>
<td>BALB/c nude mouse</td>
<td>NU</td>
<td>T</td>
<td>Shows reduced NK function</td>
</tr>
<tr>
<td>B6NU</td>
<td>B6 nude mouse</td>
<td>NU</td>
<td>T</td>
<td></td>
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<tr>
<td>NCRNU</td>
<td>NCr nude mouse</td>
<td>NU</td>
<td>T</td>
<td></td>
</tr>
<tr>
<td>NMRINU</td>
<td>NMRI nude mouse</td>
<td>NU</td>
<td>T</td>
<td></td>
</tr>
<tr>
<td>NIHNU</td>
<td>NIH nude rat</td>
<td>HI, NU, A, NW</td>
<td>T</td>
<td></td>
</tr>
<tr>
<td>9066*</td>
<td>HRN™ nude mouse</td>
<td>NU</td>
<td>T</td>
<td></td>
</tr>
<tr>
<td>CB17SC</td>
<td>C.B-17 scid mouse</td>
<td></td>
<td>T</td>
<td>B</td>
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<tr>
<td>ICRSC</td>
<td>ICR scid mouse</td>
<td></td>
<td>T</td>
<td>B</td>
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<tr>
<td>NODSC</td>
<td>NOD scid mouse</td>
<td></td>
<td>T</td>
<td>B</td>
</tr>
<tr>
<td>RAG2</td>
<td>Rag2 (129S6) mouse</td>
<td></td>
<td>T</td>
<td>B</td>
</tr>
<tr>
<td>461</td>
<td>Rag2 (B6.SJL) mouse</td>
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<td>T</td>
<td>B</td>
</tr>
<tr>
<td>601</td>
<td>Rag2 (BALB/c) mouse</td>
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<td>RAGN12</td>
<td>Rag2 (C57BL/6) mouse</td>
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<td>CBSCBG</td>
<td>Scid-beige mouse</td>
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<td>T</td>
<td>B</td>
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<tr>
<td>4111</td>
<td>Rag2/Il2rg Double Knockout Mouse</td>
<td></td>
<td>T</td>
<td>B</td>
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<tr>
<td>11503</td>
<td>CIEA BRG mouse</td>
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<td>T</td>
<td>B</td>
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<tr>
<td>NOG</td>
<td>CIEA NOG mouse®</td>
<td></td>
<td>T</td>
<td>B</td>
</tr>
</tbody>
</table>

**KEY:**

- **COAT COLOR**
  - Black and white nude
  - Black nude
  - Albino nude
  - Albino
  - White-bellied agouti

- **CELL DEFICIENCIES**
  - T Cell Deficient
  - B Cell Deficient
  - NK Cell Deficient

* Lacks P450 activity in liver

**Reduced complement activity, dysfunctional macrophages and dendritic cells, deficiencies in immune signaling, including cytokine production. The most immune deficient mouse available.**
CUSTOM BREEDING SOLUTIONS

Taconic’s fully integrated custom breeding solutions help bring novel oncology models from concept to study-ready cohorts with unprecedented speed and transparency. Customers can also combine Taconic’s portfolio of spontaneous and conditional tumor models with their existing or newly generated lines to delve into new frontiers. Our internal teams are led by PhD-scientists trained in project management principles to balance speed, cost, and quality in order to meet your customized project goals. Custom Breeding Solutions coordinates flexible tools and advanced technologies, including:

• Embryology
• Animal housing
• Molecular analysis
• Surgery and specimen collection
• Shipping animals with choice of animal identification system pre-applied

TACONIC CUSTOM BREEDING SOLUTIONS

1. Portfolio and Project Manager
2. Access to Subject Matter Expertise
3. Flexibility in Capabilities and Service Offerings
4. Global E-Business Suite and Electronic Offerings
Taconic’s GEMs Design Solutions empower our clients to develop research models specifically suited to the unique discovery study needs or therapeutic programs. As a market leader with decades of experience in custom model generation, Taconic partners with clients to design, develop, and breed high quality genetically engineered mouse and rat models.

**GENE FUNCTION STUDIES**

- **Constitutive knock out**
- **Conditional knock out**
- **Conditional knock out with Cre-ER gene switch**
- **Constitutive with the option for conditional knock out**

**TRANSGENE EXPRESSION**

- **Targeted Transgenesis**
- **Conditional Targeted Transgenesis**
- **Random Transgenesis**

**INDUCIBLE/REVERSIBLE CONTROL OF GENE FUNCTION**

- **Inducible/reversible RNA interference**
- **Inducible/reversible miRNA overexpression**

**DISEASE MODELING: MECHANISM OF PATHOGENESIS**

- **Constitutive knock in**
- **Targeted knock in**
- **Random knock in with Cre-ER gene switch**
- **Constitutive with the option for conditional knock out**

**DISEASE MODELING: DRUG TESTING & DEVELOPMENT**

- **Constitutive knock in**
- **Human gene knock in**
- **Human gene knock in with optional conditional knock out**
- **Conditional knock in**
- **Constitutive knock in point mutation with conditional knock out**
- **CRISPR Gene Editing**

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**INTEGRATED CUSTOM MODEL GENERATION AND BREEDING SOLUTIONS**

**CUSTOM MODEL GENERATION SOLUTIONS**

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ONCOLOGY

CHOOSE TACONIC
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YOUR COLLABORATIVE PARTNER
As a full-service biosciences company, Taconic can help you acquire, test, develop, breed, cryopreserve, prepare, and distribute highly relevant research lines worldwide. Whether you require custom genetically engineered, cell or tissue engrafted models or traditional models, Taconic’s scientists will partner with you to rapidly and efficiently deliver the highest quality models.

TALK TO A SCIENTIST
Our scientific teams are happy to meet and talk with you about the most efficient way to achieve your study goals. Working in partnership with clients the world over, our scientific teams offer expert advice that can help you speed up your research and reduce your overall costs.

TALK TO A REPRESENTATIVE
For general information, you can talk to a member of our customer service team. Our customer service team is here to help you make the right decisions and get the models you need fast. Contact us at info@taconic.com

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GEMS DESIGN
Taconic Biosciences GEMS Design empowers our clients to develop research models specifically suited to the unique needs of their discovery and development studies or therapeutic programs.
• Gene Inactivation
• Gene Mutation or Replacement
• CRISPR Gene Editing
• Transgene Expression
• miRNA Expression
• Cohort Production Packages

PRECISION RESEARCH MODELS
Research organizations demand precision tools that better reflect human physiology. Taconic Biosciences leads the field delivering innovative solutions to meet these continually evolving needs. Our core competencies include the delivery of complex strategies that both integrate human genetic sequences and engraft human cells and tissues into custom mouse and rat models.
• Human Gene Replacement
• Human Cell and Tissue Engraftment

GEMS MANAGEMENT
Taconic’s fully integrated GEMS Management brings innovative models from design to study-ready cohorts with unprecedented speed and transparency.
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• Rapid Colony Expansion
• Contract Breeding
• Surgical Services
• Tissue Collection
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YOUR COLLABORATIVE PARTNER
As a full-service biosciences company, Taconic can help you acquire, test, develop, breed, cryopreserve, prepare, and distribute highly relevant research lines worldwide. Whether you require custom genetically engineered, cell or tissue engrafted models or traditional models, Taconic’s scientists will partner with you to rapidly and efficiently deliver the highest quality models.

TALK TO A SCIENTIST
Our scientific teams are happy to meet and talk with you about the most efficient way to achieve your study goals. Working in partnership with clients the world over, our scientific teams offer expert advice that can help you speed up your research and reduce your overall costs.

TALK TO A REPRESENTATIVE
For general information, you can talk to a member of our customer service team. Our customer service team is here to help you make the right decisions and get the models you need fast. Contact us at info@taconic.com

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