



# SCID Models

The *scid* (severe combined immunodeficiency) mutation was discovered in a C.B-17/lcr congenic strain in 1980 by Dr. M.J. Bosma at the Fox Chase Cancer Center (Philadelphia, PA). Envigo SCID models are produced with flexible-film isolators and monitored for microbiologic integrity. SCID mice accept xenografts, making them a useful model for oncology, immunology, HIV pathology, and other fields of biomedical research.

## Envigo SCID Mice

### Research Use

- + Xenograft transplantation (1,4,6,7,8,10,11, 13,14,16,20,22,24,25,27,30,32,34,35,37)
- + Spontaneous tumors (36)
- + Cancer cell tumorigenesis (10,12,26,29,36)
- + Tumor angiogenesis (14,18,32)
- + Tumor metastatic potential (3,12,13,14,18,30,31,32,37)
- + Tumor suppression therapy (1,2,5,6,7,11,16,17,19,21,22,24,25,29,31,34,35)
- + Carcinogenesis regulation (20,23,33)
- + Tumor imaging (9)

**BALB/cJHan<sup>®</sup>Hsd-Prkdc<sup>scid</sup>**

**C.B-17/lcrHan<sup>®</sup>Hsd-Prkdc<sup>scid</sup>**

**C.B-17/lcrHsd-Prkdc<sup>scid</sup>**

### Model Characteristics

- Autosomal recessive, single nucleotide polymorphism within *Prkdc* gene on chromosome 16
- Severe combined immunodeficiency affecting T- and B-cell development
- Normal population and function of Natural Killer (NK), macrophage and granulocyte cells
- Incidence of "leaky" phenotype (spontaneous development of functional T- and B-lymphocytes) increases with age in some stocks and strains

**C.B-17/lcrHsd-Prkdc<sup>scid</sup> Lyst<sup>bg-J</sup>**

### Model Characteristics

- Autosomal recessive, single nucleotide polymorphism within *Prkdc* gene on chromosome 16
- Autosomal recessive *beige* (*bg-J*) mutation on chromosome 13
- Diminished Natural Killer (NK) cell activity relative to other SCID models
- Severe combined immunodeficiency affecting T- and B-cell development
- Severe lymphopenia
- "Leaky" phenotype significantly suppressed relative to other SCID models

**NOD.CB17-Prkdc<sup>scid</sup>/NCrHsd**

### Model Characteristics

- *Prkdc<sup>scid</sup>* mutation has been transferred onto a Non-Obese Diabetic mouse background
- Severe combined affecting T- and B-cell development
- Functional in Natural Killer (NK) cells and APC cells
- Absence of circulating complement
- High incidence of lethal thymic lymphomas
- Does not show signs of autoimmune diabetes
- Leakiness increase with age is minimal



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