

The term “inflammatory bowel disease” (IBD) commonly refers to two chronic conditions that involve inflammation of the intestines: ulcerative colitis and Crohn’s disease. The two conditions share a number of common characteristics, but have notable differences. Ulcerative colitis is an inflammatory disease that primarily affects the mucosal lining of the large intestine (colon) and rectum. Crohn’s disease may involve any part of the gastro-intestinal tract, but commonly affects the last part of the small intestine and portions of the large intestine. Inflammation in Crohn’s disease extends more deeply into the intestinal wall.

A combination of genetic characteristics and enteric environment (microbiota) are believed to contribute to the susceptibility, initiation and progression of these diseases in both man and mouse. IBD is a clinically and genetically heterogeneous disorder with several genetic loci shown to contribute to increased susceptibility (Cho, 2004; Duerr, 2002; Cho, 2000; Brodmerkel, 2003; Jurjus, 2004). Among these are Crohn’s Disease-predisposing variants in the *NOD2/CARD15* gene (Ogura, 2001; Hugot, 2001) associated with intracellular sensing of a muramyl dipeptide ligand. The genetic heterogeneity of IBD syndromes in humans is reinforced in mouse models by the finding that IBD can be elicited by disruption of a variety of genes associated either with immune responsiveness or maintenance of mucosal integrity. The Jackson Laboratory offers a number of mouse models that may be used to study various features of inflammatory bowel disease. The following list briefly summarizes these strains.

Because IBD development in many of the mouse models requires interaction between the host genome and undefined microbial agents in the colon, conventional SPF (Specific Pathogen-Free) housing instead of full-barrier SPF is often required for full development of IBD. The mice maintained in the Production colonies at The Jackson Laboratory are maintained in full-barrier

facilities. Mice that would otherwise develop spontaneous colitic lesions will likely have delayed onset with mild symptoms or no onset and also be devoid of a multitude of pathogenic/opportunistic microbiota (such as *Helicobacter hepaticus* and *Klebsiella oxytoca*). Although these particular commensal bacteria do not appear to be the colitogenic “triggers” in immune-competent mice, they serve as markers for the as yet unknown colitogenic agents. Presentation of specific clinical features in full barrier-bred juvenile mice carrying targeted mutations may require exposure to microbes in soiled bedding from colitis-susceptible mice bred under standard SPF. Colitis onset and severity are variable with many of these IBD-susceptible stocks. Individual animals from a given colony can be expected to show some variability in rate and severity of lesion development.

Whereas colitis induced by targeted mutations generally is very sensitive to the nature of the enteric flora, both barrier and non-barrier mice will respond well to experimentally-induced colitis [e.g., by administration of dextran sodium sulfate (DSS) in drinking water (Mahler, 1998) or by rectal administration of trinitrobenzene sulfonic acid (TNBS) (Neurath, 1995)]. However, inbred strain background is an important factor regardless of the model chosen. Some backgrounds confer greater colitis susceptibility than others (i.e., IL10-deficient C3H/HeJBir mice demonstrate early and severe colitic lesions in cecum and large intestine, while IL10-deficient C57BL/6J (B6) mice have delayed age of onset and lesion severity is greatly diminished (Mahler, 1998; Mahler and Leiter, 2002; Beckwith, 2005)). Similarly, C3H/HeJBir wild-type mice are significantly more sensitive to the colitogenic effects of DSS or TNBS than are B6 mice. As The Jackson Laboratory acquires new colitis models on various inbred strain backgrounds for distribution, the published literature serves as a guide but the user should expect changes in the published phenotype mediated by any conditions unique to a given animal facility.

References

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IBD Models

(see referenced page for full strain detail)

Card15 Strain

| page | stock number | strain |
|------|--------------|--------------------------------------|
| 4 | 005763 | B6.129S1-Card15 ^{tm1Flv} /J |

Cftr Strain

| page | stock number | strain |
|------|--------------|------------------------------------|
| 4 | 002515 | B6.129S6-Cftr ^{tm1Kth} /J |

HSV-Tk Strain

| page | stock number | strain |
|------|--------------|---------------------------|
| 4 | 005698 | B6.Cg-Tg(Gfap-Tk)7.1Mvs/J |

Il2 Strains

| page | stock number | strain |
|------|--------------|---------------------------------------|
| 5 | 002252 | B6.129P2-Il2 ^{tm1Hor} /J |
| 5 | 002229 | C.129P2(B6)-Il2 ^{tm1Hor} /J |
| 5 | 002228 | C3.129P2(B6)-Il2 ^{tm1Hor} /J |

Il2ra Strains

| page | stock number | strain |
|------|--------------|------------------------------------|
| 5 | 002462 | B6;129S4-Il2ra ^{tm1Dw} /J |
| 6 | 002952 | B6.129S4-Il2ra ^{tm1Dw} /J |

Il2rg Strains

| page | stock number | strain |
|------|--------------|-------------------------------------|
| 6 | 003174 | B6.129S4-Il2rg ^{tm1Wjl} /J |
| 6 | 003169 | C.129S4-Il2rg ^{tm1Wjl} /J |

Il10 Strains

| page | stock number | strain |
|------|--------------|--|
| 6 | 004368 | 129-Il10 ^{tm1Cgn} /J |
| 7 | 002251 | B6.129P2-Il10 ^{tm1Cgn} /J |
| 7 | 002250 | B10.129P2(B6)-Il10 ^{tm1Cgn} /J |
| 7 | 004333 | C.129P2(B6)-Il10 ^{tm1Cgn} /J |
| 7 | 004326 | C3Bir.129P2(B6)-Il10 ^{tm1Cgn} /Lt |
| 8 | 003968 | C3Bir.129P2(B6)-Il10 ^{tm1Cgn} /J |
| 8 | 004328 | NOD.Cg-H2nb1 Il10 ^{tm1Cgn} /Lt |
| 8 | 004266 | NOD.Cg-Il10 ^{tm1Cgn} /DvsJ |
| 8 | 004291 | NOD.Cg-Il10 ^{tm1Cgn} Il4 ^{tm1Cgn} /DvsJ |
| 9 | 004982 | C3Bir.Cg-Il10 ^{tm1Cgn} (D3Mit11-D3Mit11-D3Mit19)/Lt |
| 9 | 005913 | B6.Cg-Il10 ^{tm1Cgn} (D3Mit11-D3Mit19)/Lt |

Il10rb Strain

| page | stock number | strain |
|------|--------------|--------------------------------------|
| 9 | 005027 | B6.129S2-Il10rb ^{tm1Agt} /J |

Lyzs Strain

| page | stock number | strain |
|------|--------------|---|
| 9 | 004781 | B6.129P2-Lyzs ^{tm1(cre)lfo} /J |

Nfkb1 Strain

| page | stock number | strain |
|------|--------------|--------------------------------------|
| 10 | 002849 | BB6;129P2-Nfkb1 ^{tm1Bal} /J |

Nos2 Strains

| page | stock number | strain |
|------|--------------|------------------------------------|
| 10 | 002596 | B6;129P2-Nos2 ^{tm1Lau} /J |
| 10 | 002609 | B6.129P2-Nos2 ^{tm1Lau} /J |

Tbx21 Strain

| page | stock number | strain |
|------|--------------|-------------------------------------|
| 11 | 004648 | B6.129S6-Tbx21 ^{tm1Glm} /J |

Tcra, Tcrb, Tcrd Strains

| page | stock number | strain |
|------|--------------|---|
| 11 | 002115 | B6;129S2-Tcra ^{tm1Mom} /J |
| 11 | 002116 | B6.129S2-Tcra ^{tm1Mom} /J |
| 12 | 004364 | C.Cg-Tcra ^{tm1Mom} Tcrb ^{tm1Mom} /J |
| 12 | 002117 | B6;129P2-Tcrb ^{tm1Mom} /J |
| 12 | 002118 | B6.129P2-Tcrb ^{tm1Mom} /J |
| 13 | 002121 | B6;129P-Tcrb ^{tm1Mom} Tcrd ^{tm1Mom} /J |
| 13 | 002122 | B6.129P2-Tcrb ^{tm1Mom} Tcrd ^{tm1Mom} /J |
| 13 | 002119 | B6;129P2-Tcrd ^{tm1Mom} /J |
| 13 | 002120 | B6.129P2-Tcrd ^{tm1Mom} /J |

Was Strain

| page | stock number | strain |
|------|--------------|---------------------------|
| 14 | 003292 | 129S6/SvEvTac-Wastm1Sbs/J |

Web Resources

Research Models for Inflammatory Bowel Disease

www.jax.org/jaxmice/models/immunology

Links to research models in various disease areas, information on upcoming Courses and Conferences, information on current research at The Jackson Lab and links to other resources to support research models for immunology and inflammation.

JAX® Mice Database

www.jax.org/jaxmice/search

With information on over 2800 strains of laboratory mice, view strain data sheets, genotyping protocols, current pricing and availability. Search by research application or enter the stock number of the strain of interest and view the Strain Data Sheet.

Newly Available Models Recently Released for Distribution

www.jax.org/jaxmice/newstrains

The supply of mice from strains that have recently been released for distribution may be limited. Colony sizes are ultimately sized based on the broad needs of the research community. Please refer to the JAX® Mice Database for current availability and price information. If your experiments require numbers of mice that exceed our current supply, we will work with you to meet your needs (please contact our Customer Service Department or email JAX® Services at jaxservices@jax.org).

New Strains Under Development, Not Yet Available

www.jax.org/jaxmice/interestlist

The Jackson Laboratory serves as a worldwide distributor and national repository for common and rare strains of inbred mice and mice carrying spontaneous mutations or induced mutations (*i.e.*, transgenic, targeted/"knockout", or chemically induced mutations). At any one time, we have over 100 strains at various stages of development and colony expansion. We encourage you to register with us if you are interested in any strain that is under development or on hold. Registering interest allows you to: 1) Obtain advance notification of strain availability and opportunity to order prior to the strain being published as available; 2) Provide input affecting speed and quantity of availability.

Email Notifications

www.jax.org/jaxmice/subscribe

Register for email notifications and updates on new research models.

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|---|---|
| Symbol <i>Card15^{tm1Flv}</i> | Gene Name caspase recruitment domain family, member 15 |
| | Allele Name targeted mutation 1, Richard A Flavell |
| | Symbol Common Name(s) F830032C23Rik; IBD1; Nod2; Nod2- |
| Strain Name | B6.129S1-<i>Card15^{tm1Flv}/J</i> |
| Stock Number | 005763 |
| Application(s) | CD Antigens, Antigen Receptors, and Histocompatibility Markers (genes regulating susceptibility to infectious disease and endotoxin); Immunodeficiency (Inflammatory bowel disease) (Tlr deficiency) (defects in humoral immune responses); Inflammation (Inflammatory bowel disease) (Tlr deficiency); Intracellular Signaling Molecules; Research Tools: Immunology and Inflammation Research (Macrophage Deficiency) |
| Additional Research Areas | Cell Biology Research; Hematological Research; Research Tools |
| Phenotype | Homozygous mice are viable and fertile with normal lymphoid and myeloid cellularity and no intestinal inflammation up to 6 months of age. Homozygotes do not express the targeted gene in spleen or intestinal crypts. Null mice, as well as antigen presenting cells derived from them, lack the protective immunity (IgG1, interleukin-6, and NF-kappaB-related responses) normally afforded by endogenous protein recognition of its ligand, bacterial muramyl dipeptide (MDP). Mice homozygous for the mutation have increased susceptibility to oral (intra-gastric) bacterial challenge and diminished cryptdins. This mouse may be useful in studies of Crohn's disease and other inflammatory bowel diseases, innate immunity, signal transduction, and bacterial susceptibility. |
| Primary Reference | Kobayashi KS, Chamaillard M, Ogura Y, Henegariu O, Inohara N, Nunez G, Flavell RA. 2005. Nod2-dependent regulation of innate and adaptive immunity in the intestinal tract. <i>Science</i> 307:731-4. |

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| Symbol <i>Cftr^{tm1Rol}</i> | Gene Name cystic fibrosis transmembrane conductance regulator homolog |
| | Allele Name targeted mutation 1, Kirk R Thomas |
| | Symbol Common Name(s) 508; Abcc7 |
| Strain Name | B6.129S6-<i>Cftr^{tm1Kth}/J</i> |
| Stock Number | 002515 |
| Application(s) | Inflammation (Inflammatory bowel disease) |
| Additional Research Areas | Metabolism Research; Mouse/Human Gene Homologs |
| Phenotype | The <i>Cftr^{tm1Kth}</i> targeted mutation corresponds to the delta 508 mutation in humans. Homozygous mutant mice show an increased mortality within the first month after birth, with ~60% mortality by post-weaning. Those that survive are fertile, but females are poor breeders. Mutant mice are also reduced in size compared to normal wildtype mice. Those that do not survive to adulthood show bowel obstructions, bowel strictures and peritonitis. Lungs, pancreas, gall bladder, male reproductive tract, lacrimal gland, and submandibular glands from homozygous mice appear normal regardless of the survival of the animal. Homozygous mice also show a tissue-specific loss of CFTR transcripts in the intestine. |
| Primary Reference | Zeiber BG, Eichwald E, Zabner J, Smith JJ, Puga AP, McCray PB Jr., Capecchi MR, Welsh MJ, Thomas KR. 1995. A mouse model for the delta F508 allele of cystic fibrosis. <i>J Clin Invest</i> 96:2051-2064. |

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| Symbol <i>HSV-Tk</i> | Gene Name herpes simplex virus thymidine kinase |
| Strain Name | B6.Cg-Tg(Gfap-Tk)7.1Mvs/J |
| | Strain Former & Common Name(s): GFAP-TK; Gfap-HSV-Tk |
| | Promoter: <i>Gfap</i> , glial fibrillary acidic protein, mouse |
| Stock Number | 005698 |
| Application(s) | Immunodeficiency (Inflammatory bowel disease); Inflammation (Inflammatory bowel disease) |
| Additional Research Areas | Internal/Organ Research; Research Tools: Genetics Research; Neurobiology Research |

Phenotype Mice hemizygous for the transgenic insert are viable, normal in size, and do not display any behavioral abnormalities. Proliferating cells that express the herpes simplex virus thymidine kinase (HSV-TK) transgene will metabolize ganciclovir (GCV) to toxic nucleotide analogues and undergo cell death. Transgenic males are infertile. Coexpression of endogenous *Gfap* and transgenic HSV-*Tk* occurs in brain astrocytes and adult neural stem cells, enteric glia, hepatic stellate cells, and unknown cells in heart, lung, kidney, adrenal, and spleen. Chronic ganciclovir treatment for 21 days depletes GFAP-positive adult neural stem cells from forebrain proliferative zones. Stab or spinal cord injury with high dose ganciclovir treatment for 7 days induces reactive astrocyte cell death leading to altered leukocyte trafficking and impaired injury healing. High dose ganciclovir treatment for 14 days ablates GFAP-positive glia from the jejunum and ileum leading to fulminant and fatal jejuno-ileitis. This mutant can be used in studies of adult neurogenesis, nervous system injury/repair mechanisms, and inflammatory bowel disease.

Primary Reference Schorle H, Holtschke T, Hunig T, Schimpl A, Horak I. 1991. Development and function of T cells in mice rendered interleukin-2 deficient by gene targeting. *Nature* 352 :621-24.

Symbol
Il2tm1Cgn

Gene Name **interleukin 2**
Allele Name **targeted mutation 1, Ivan Horak**
Symbol Common Name(s) **Il-2**

Strain Name **B6.129P2-*Il2tm1Hor*/J**
Strain Former & Common Name(s): C57BL/6J-*Il2tm1Hor*; B6.129P2-*Il2tm1Hor*

Stock Number **002252**

Strain Name **C.129P2(B6)-*Il2tm1Hor*/J**
Strain Former & Common Name(s): BALB/c-*Il2tm1Hor*; C.129P2(B6)-*Il2tm1Hor*

Stock Number **002229**

Strain Name **C3.129P2(B6)-*Il2tm1Hor*/J**
Strain Former & Common Name(s): C3H/HeJCrIBR-*Il2tm1Hor*; C3.129P2(B6)-*Il2tm1Hor*

Stock Number **002228**

Application(s) Autoimmunity (hemolytic anemia); Growth Factors/Receptors/Cytokines; Inflammation (Inflammatory bowel disease)

Additional Research Areas Cancer Research; Hematological Research

Phenotype Homozygous mutant mice for the *Il2tm1Hor* targeted mutation show no apparent deficit in thymocyte differentiation or selection, types and numbers of T cells from spleens and lymph nodes are comparable to those of wildtype. They do have an impaired response to polyclonal T cell activators in the absence of additional IL2, deficits in their helper function and a reduction in natural killer cell activity. There is significant pre-weaning and post-weaning loss of homozygotes on the C3H/HeJCrIBR and C57BL/6J genetic backgrounds. In addition, homozygous mice develop inflammatory bowel disease between 6 and 15 weeks of age and reportedly die within 10-25 weeks under conventional housing conditions. Homozygotes on the C57BL/6J genetic background show an atrophied pancreas with apparently intact islets. Homozygotes on the BALB/c genetic background do not develop inflammatory bowel disease symptoms but rather die 3-5 weeks postnatally of a form of hemolytic anemia. For a more detailed description please refer to the JAX Notes Fall 1996 article (www.jax.org/jaxmice/library/notes/467a).

Primary Reference Schorle H, Holtschke T, Hunig T, Schimpl A, Horak I. 1991. Development and function of T cells in mice rendered interleukin-2 deficient by gene targeting. *Nature* 352 :621-24.

Symbol
Il2ra^{tm1Dw}

Gene Name **interleukin 2 receptor, alpha chain**
Allele Name **targeted mutation 1, Dennis M Willerford**

Strain Name **B6;129S4-*Il2ra^{tm1Dw}*/J**
Strain Former & Common Name(s): B6;129S-*Il2ra^{tm1Dw}*; B6;129S4-*Il2ra^{tm1Dw}*

Stock Number **002462**

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|---------------------------|---|
| Strain Name | B6.129S4-<i>Il2ra</i>^{tm1Dw}/J |
| Stock Number | 002952 |
| Application(s) | Autoimmunity (hemolytic anemia); Growth Factors/Receptors/Cytokines; Immunodeficiency (Inflammatory bowel disease); Inflammation (Inflammatory bowel disease) |
| Additional Research Areas | Cancer Research |
| Phenotype | Mice homozygous for the <i>Il2ra</i> ^{tm1Dw} targeted mutation are characterized by a lymphoproliferative disorder, hemolytic anemia, and an inflammatory bowel disease beginning at approximately 9 weeks of age; also known as p55 gene chain, <i>Ly43</i> , CD25. |
| Primary Reference | Willerford DM, Chen J, Ferry JA, Davidson L, Ma A, Alt FW. 1995. Interleukin-2 receptor alpha chain regulates the size and content of the peripheral lymphoid compartment. <i>Immunity</i> 3:521-530. |

Symbol
***Il2rg*^{tm1Wjl}**

Gene Name **interleukin 2 receptor, gamma chain**
Allele Name **targeted mutation 1, Warren J Leonard**

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| Strain Name | B6.129S4-<i>Il2rg</i>^{tm1Wjl}/J |
| Stock Number | 003174 |
| Strain Name | C.129S4-<i>Il2rg</i>^{tm1Wjl}/J |
| Stock Number | 003169 |
| General Terms & Conditions | PHS/NIH REQUIRES THAT FOR-PROFIT ENTITIES MUST OBTAIN A USE LICENSE BEFORE PURCHASING THESE MICE FROM THE JACKSON LABORATORY. www.jax.org/jaxmice/orders/legalletter/002881_legalletter |
| Application(s) | Growth Factors/Receptors/Cytokines; Inflammation (Inflammatory bowel disease); Research Tools: Immunology and Inflammation Research (NK Cell Deficiency) |
| Additional Research Areas | Cancer Research |
| Phenotype | Mice homozygous for the <i>Il2rg</i> ^{tm1Wjl} targeted mutation are viable and fertile. Mutant mice have hypoplastic thymuses with a 10-fold reduction in the absolute number of lymphocytes. They have a limited number of mature splenic B and T cells, lack NK cells and Peyer's patches, and development of gut-associated intraepithelial lymphocytes is diminished. <i>In vitro</i> studies show defects in NK activity, IL4-directed Ig class switching of thymocytes, and thymocyte mitogenic responses. |
| Primary Reference | Cao X, Shores EW, Hu-Li J, Anver MR, Kelsall BL, Russell SM, Drago J, Noguchi M, Grinberg A, Bloom ET, Paul WE, Katz SI, Love PE, Leonard WJ. 1995. Defective lymphoid development in mice lacking expression of the common cytokine receptor gamma chain. <i>Immunity</i> 2:223-38. |

Symbol
***Il10*^{tm1Cgn}**

Gene Name **interleukin 10**
Allele Name **targeted mutation 1, University of Cologne**

For all *Il10*^{tm1Cgn} strains:

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| General Terms & Conditions | Notice regarding mice developed by the University of Cologne. www.jax.org/jaxmice/orders/legalletter/muellerlegalletter |
| Application(s) | Autoimmunity; Growth Factors/Receptors/ Cytokines; Immunodeficiency; Inflammation (Inflammatory bowel disease) |
| Additional Research Areas | Cancer Research; Hematological Research |
| Strain Name | 129-<i>Il10</i>^{tm1Cgn}/J |
| Stock Number | 004368 |

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| Add'l General Terms & Cond. | OncoMouse™ requires a license from DuPont, see Licenses for Strains with OncoMouse™ Technology . |
| Phenotype | Mice homozygous for the targeted mutation are viable and fertile when housed under SPF conditions. At 3 months of age mutant mice exhibit severe intestinal lesions, increased granulocyte counts and elevated serum amyloid A levels. There is a 67% prevalence of colorectal adenocarcinoma. This mutant mouse strain represents a model that may be useful in studies related to inflammatory bowel disease. |
| Primary Reference | Berg DJ, Davidson N, Kuhn R, Muller W, Menon S, Holland G, Thompson-Snipes L, Leach MW, Rennick D. 1996. Enterocolitis and colon cancer in interleukin-10-deficient mice are associated with aberrant cytokine production and CD4(+) TH1-like responses. <i>J Clin Invest</i> 98:1010-1020. |
| Strain Name | B6.129P2-<i>Il10^{tm1Cgn}/J</i> |
| Stock Number | Strain Former & Common Name(s): B6.129P2- <i>Il10^{tm1Cgn}</i> ; C57BL/6- <i>Il10^{tm1Cgn}</i> 002251 |
| Phenotype | Mice homozygous for the <i>Il10^{tm1Cgn}</i> targeted mutation are viable and fertile when housed under SPF conditions. Under conventional housing conditions homozygous mutant mice are small in size, anemic, and have chronic enterocolitis. Intestinal problems are far less severe (local inflammation of the proximal colon) in a pathogen-free environment. Lymphocyte development and antibody responses are normal in homozygous mutant mice. For a more detailed description please refer to the JAX Notes Fall 1997 article (www.jax.org/jaxmice/library/notes/471a). |
| Primary Reference | Kuhn R, Lohler J, Rennick D, Rajewsky K, Muller W. 1993. Interleukin-10-deficient mice develop chronic enterocolitis. <i>Cell</i> 75:263-274. |
| Strain Name | B10.129P2(B6)-<i>Il10^{tm1Cgn}/J</i> |
| Stock Number | Strain Former & Common Name(s): C57BL/10- <i>Il10^{tm1Cgn}</i> ; B10.129P2(B6)- <i>Il10^{tm1Cgn}</i> 002250 |
| Phenotype | Mice homozygous for the <i>Il10^{tm1Cgn}</i> targeted mutation are viable and fertile when housed under SPF conditions. Under conventional housing conditions homozygous mutant mice are small in size, anemic, and have chronic enterocolitis. Intestinal problems are far less severe (local inflammation of the proximal colon) in a pathogen-free environment. Lymphocyte development and antibody responses are normal in homozygous mutant mice. For a more detailed description please refer to the JAX Notes Fall 1997 article (www.jax.org/jaxmice/library/notes/471a). |
| Primary Reference | Kuhn R, Lohler J, Rennick D, Rajewsky K, Muller W. 1993. Interleukin-10-deficient mice develop chronic enterocolitis. <i>Cell</i> 75:263-274. |
| Strain Name | C.129P2(B6)-<i>Il10^{tm1Cgn}/J</i> |
| Stock Number | Strain Former & Common Name(s): C.129P2(B6)- <i>Il10^{tm1Cgn}</i> 004333 |
| Add'l General Terms & Cond. | OncoMouse™ requires a license from DuPont, see Licenses for Strains with OncoMouse™ Technology . |
| Additional Research Areas | Cell Biology Research; Internal/Organ Research |
| Phenotype | Mice homozygous for the <i>Il10^{tm1Cgn}</i> targeted mutation are viable and fertile when housed under specific pathogen-free (SPF) conditions. The phenotype of <i>Il10</i> -deficient mice is strongly influenced by the genetic background. At 3 months of age, <i>Il10^{tm1Cgn}</i> homozygous mutant mice on a BALB/c background exhibit severe intestinal lesions, increased granulocyte counts, and elevated serum amyloid A levels. There is a 29% prevalence of colorectal adenocarcinoma. This mutant mouse strain represents a model that may be useful in studies related to inflammatory bowel disease. |
| Primary Reference | Berg DJ, Davidson N, Kuhn R, Muller W, Menon S, Holland G, Thompson-Snipes L, Leach MW, Rennick D. 1996. Enterocolitis and colon cancer in interleukin-10-deficient mice are associated with aberrant cytokine production and CD4(+) TH1-like responses. <i>J Clin Invest</i> 98:1010-1020. |
| Strain Name | C3Bir.129P2(B6)-<i>Il10^{tm1Cgn}/Lt</i> |
| Stock Number | Important Note: This strain is homozygous for <i>Pde6b^{rd1}</i> and <i>Tlr4^{Lps-d}</i> . Also, this strain is probably homozygous for a chromosomal inversion on Chromosome 6, as are mice of the background strain, C3H/HeJBir. See JAX Notes Issue #491, Fall 2003, p. 15 (www.jax.org/jaxmice/library/notes/491a). 004326 |
| Phenotype | Mice homozygous for the <i>Il10^{tm1Cgn}</i> targeted mutation are viable and fertile when housed under SPF conditions. Under conventional housing conditions homozygous mutant mice are small in size, anemic, |

and have chronic enterocolitis. It should be noted that the phenotype of cecocolitis in this strain requires an interaction with an as yet undefined component of the enteric flora. Helicobacters do not appear to be the environmental triggers. Intestinal problems are far less severe (local inflammation of the proximal colon) in a pathogen-free environment. Lymphocyte development and antibody responses are normal in homozygous mutant mice. For a more detailed description please refer to the Fall 1997 JAX Notes article (www.jax.org/jaxmice/library/notes/471a).

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|---------------------------|---|
| Primary Reference | Lytle C, Tod TJ, Vo KT, Lee JW, Atkinson RD, Straus DS. 2005. The peroxisome proliferator-activated receptor gamma ligand rosiglitazone delays the onset of inflammatory bowel disease in mice with interleukin 10 deficiency. <i>Inflamm Bowel Dis</i> 11:231-43. |
| Strain Name | C3Bir.129P2(B6)-<i>Il10^{tm1Cgn}/J</i> Strain Former & Common Name(s): C3Bir.129P2(B6)- <i>Il10^{tm1Cgn}</i> Important Note: This strain is homozygous for <i>Pde6b^{rd1}</i> and <i>Tlr4^{Lps-d}</i> . Also, this strain is probably homozygous for a chromosomal inversion on Chromosome 6, as are mice of the background strain, C3H/HeJBir. See JAX Notes Issue #491, Fall 2003, p. 15 (www.jax.org/jaxmice/library/notes/491a). |
| Stock Number | 003968 |
| Phenotype | Mice homozygous for the <i>Il10^{tm1Cgn}</i> targeted mutation are viable and fertile when housed under SPF conditions. Under conventional housing conditions homozygous mutant mice are small in size, anemic, and have chronic enterocolitis. It should be noted that the phenotype of cecocolitis in this strain requires an interaction with an as yet undefined component of the enteric flora. Helicobacters do not appear to be the environmental triggers. Intestinal problems are far less severe (local inflammation of the proximal colon) in a pathogen-free environment. Lymphocyte development and antibody responses are normal in homozygous mutant mice. For a more detailed description please refer to the Fall 1997 JAX Notes article (www.jax.org/jaxmice/library/notes/471a). |
| Primary Reference | Bristol IJ, Farmer MA, Cong Y, Zheng XX, Strom TB, Elson CO, Sundberg JP, Leiter EH 2000. Heritable susceptibility for colitis in mice induced by IL-10 deficiency. <i>Inflamm Bowel Dis</i> 6:290-302. |
| Strain Name | NOD.Cg-<i>H2^{nb1} Il10^{tm1Cgn}/Lt</i> Strain Former & Common Name(s): NOD/Lt- <i>H2^{nb1} Il10^{tm1Cgn}</i> |
| Stock Number | 004328 |
| Strain Name | NOD.Cg-<i>Il10^{tm1Cgn}/DvsJ</i> |
| Stock Number | 004266 |
| Application(s) | Immunology and Inflammation Research: Immunodeficiency Associated with Other Defects; Lymphoid Tissue Defects; Vaccine Development; Research Tools: Immunology and Inflammation Research (genes regulating susceptibility to infectious disease and endotoxin) (production of T cell lines and hybridomas) (specific T cell deficiency) |
| Additional Research Areas | Diabetes and Obesity Research; Internal/Organ Research; Research Tools |
| Phenotype | Mice homozygous for the <i>Il10^{tm1Cgn}</i> targeted mutation are viable and fertile when housed under SPF conditions. This mutant develops type 1 diabetes at the same rate as the NOD/Lt parental strain. The <i>Il10^{tm1Cgn}</i> mutation also renders this NOD/Lt stock susceptible to colitis (although not as severe as other strains of <i>Il10</i> deficient mice) when maintained under standard housing conditions. |
| Primary Reference | Serreze DV, Chapman HD, Post CM, Johnson EA, Suarez-Pinzon WL, Rabinovitch A. 2001. Th1 to Th2 cytokine shifts in nonobese diabetic mice: sometimes an outcome, rather than the cause, of diabetes resistance elicited by immunostimulation. <i>J Immunol</i> 166:1352-9. |
| Strain Name | NOD.Cg-<i>Il10^{tm1Cgn} Il4^{tm1Cgn}/DvsJ</i> Strain Former & Common Name(s): NOD. <i>Il4/10^{null}</i> |
| Stock Number | 004291 |
| Additional Research Areas | Diabetes and Obesity Research |
| Phenotype | Mice homozygous for the <i>Il10</i> and <i>Il4</i> targeted mutations are viable and fertile when housed under SPF conditions. NOD/Lt mice deficient for both of these genes develop type 1 diabetes at a rate equivalent to the parental strain. The <i>Il10^{tm1Cgn}</i> mutation also renders this NOD/Lt stock susceptible to colitis (although not as severe as other strains of <i>Il10</i> deficient mice) when maintained under standard housing conditions. |

Primary Reference Serreze DV, Chapman HD, Post CM, Johnson EA, Suarez-Pinzon WL, Rabinovitch A. 2001. Th1 to Th2 cytokine shifts in nonobese diabetic mice: sometimes an outcome, rather than the cause, of diabetes resistance elicited by immunostimulation. *J Immunol* 166:1352-9.

Strain Name C3Bir.Cg-*Il10^{tm1Cgn}* (*D3Mit11-D3Mit11-D3Mit19*)/Lt

Stock Number 004982

Strain Former & Common Name(s): C3H.B6.IL10-/-; CB-R1

Strain Name B6.Cg-*Il10^{tm1Cgn}* (*D3Mit11-D3Mit19*)/Lt

Stock Number 005913

Strain Former & Common Name(s): BC-R3

Phenotype Homozygous mice are IL10-deficient and develop spontaneous colitic lesions in the cecum and large intestine as early as 6-8 weeks of age. The severity and age of onset of colitis varies with the donor type and location of the cytokine deficiency colitis susceptibility 1 (*Cdcs1*) interval. Generally, the C3H/HeJBir region encompassed by *D3Mit348* through *D3Mit254* (127.7-132.5 megabases) confers increased colitis susceptibility on the B6.129P2-*Il10^{tm1Cgn}* background. (Beckwith J, *et al.*, 2005) Mice on the B6.129P2-*Il10^{tm1Cgn}* background are prone to rectal prolapse particularly when carrying susceptibility alleles from Chromosome 3. Granulocyte populations in peripheral blood increase upon lesion development and provide a robust non-lethal assessment of colitis severity.

Selected Reference(s) Farmer MA, Sundberg JP, Bristol IJ, Churchill GA, Li R, Elson CO, Leiter EH. 2001. A major quantitative trait locus on chromosome 3 controls colitis severity in IL-10-deficient mice. *Proc Natl Acad Sci U S A* 98:13820-5. Beckwith J, Cong Y, Sundberg JP, Elson CO, Leiter EH. 2005. *Cdcs1*, a major colitogenic locus in mice, regulates innate and adaptive immune response to enteric bacterial antigens. *Gastroenterology* 129:1473-84.

Symbol
Il10rb^{tm1Agt}

Gene Name interleukin 10 receptor, beta
Allele Name targeted mutation 1, Michael Aguet

Strain Name B6.129S2-*Il10rb^{tm1Agt}*/J

Strain Former & Common Name(s): CRFB4-/-

Stock Number 005027

Application(s) Inflammation (Inflammatory bowel disease); Lymphoid Tissue Defects

Additional Research Areas Internal/Organ Research

Phenotype Mice that are homozygous for the targeted mutation are viable, fertile, normal in size and do not display any gross physical or behavioral abnormalities. No gene product (mRNA) is detected by Northern blot analysis of ES cells. After 12 weeks of age, approximately 60% of mutant mice develop chronic colitis and increased numbers of splenocytes resulting in splenomegaly. Bone marrow-derived macrophages, splenocytes and peritoneal cells derived from homozygotes do not respond to IL-10. Mutant mice housed in non-SPF conditions do not breed well. This mutant mouse strain may be useful in studies of the role of interleukin-10 in inflammatory diseases such as chronic colitis.

Primary Reference Spencer SD, Di Marco F, Hooley J, Pitts-Meek S, Bauer M, Ryan AM, Sordat B, Gibbs VC, Aguet M. 1998. The orphan receptor CRF2-4 is an essential subunit of the interleukin 10 receptor. *J Exp Med* 187:571-8.

Symbol
Lyzs^{tm1}(cre)Ifo

Gene Name lysozyme
Allele Name targeted mutation 1, Irmgard Foerster

Strain Name B6.129P2-*Lyzs^{tm1}(cre)Ifo*/J

Strain Former & Common Name(s): B6.129-*Lyzs^{tm1}(cre)Ifo*/J; B6.129P2-*Lyzs^{tm1}(cre)Ifo*; LysMcrc

Stock Number 004781

General Terms & Conditions Cre recombinase requires a license from either Dupont or Bristol Myers Squibb Company, see [Licenses for Strains using Cre-lox Technology](#).

Application(s) Autoimmunity (experimentally induced rheumatoid arthritis); CD Antigens, Antigen Receptors, and Histocompatibility Markers (genes regulating susceptibility to infectious disease and endotoxin); Growth

Factors/Receptors/Cytokines; Immunodeficiency (Neutrophil Defects); Inflammation (Neutrophil function) (Inflammatory bowel disease); Lymphoid Tissue Defects (hematopoietic development) (myeloid hyperplasia); Research Tools

Additional Research Areas

Phenotype

Developmental Biology Research; Hematological Research; Internal/Organ Research; Research Tools
This strain expresses Cre recombinase from the endogenous *Lyzs* locus. When crossed with a strain containing *loxP* site flanked sequence of interest, Cre-mediated recombination results in deletion of the targeted gene in the myeloid cell lineage, including monocytes, mature macrophages, and granulocytes. Mice that are homozygous for the targeted mutation are viable, fertile, normal in size and do not display any gross physical or behavioral abnormalities. This strain represents an effective tool for generating myeloid cell-specific targeted mutants.

Selected Reference(s)

Clausen BE, Burkhardt C, Reith W, Renkawitz R, Forster I. 1999. Conditional gene targeting in macrophages and granulocytes using *LysMcre* mice. *Transgenic Res.* 8:265-77.
Takeda K, Clausen BE, Kaisho T, Tsujimura T, Terada N, Forster I, Akira S. 1999. Enhanced Th1 activity and development of chronic enterocolitis in mice devoid of Stat3 in macrophages and neutrophils. *Immunity* 10:39-49.

Symbol

Nfkb1^{tm1Bal}

Gene Name

nuclear factor of kappa light chain gene enhancer in B-cells 1, p105

Allele Name

targeted mutation 1, David Baltimore

Strain Name

B6;129P2-*Nfkb1^{tm1Bal}/J*

Strain Former & Common Name(s): B6;129P-*Nfkb1^{tm1Bal}*; B6;129P2-*Nfkb1^{tm1Bal}*

Stock Number

002849

Application(s)

Immunodeficiency (Inflammatory bowel disease)

Phenotype

Mice homozygous for the *Nfkb1^{tm1Bal}* targeted mutation are viable and fertile. Homozygous mutant mice exhibit defective B cell responses, defective responses to infection, and also defects in basal and specific antibody production.

Primary Reference

Sha WC, Liou H, Tuomanen EI, Baltimore D. 1995. Targeted disruption of the p50 subunit of NF-kappa B leads to multifocal defects in immune responses. *Cell* 80 :321-330.

Symbol

Nos2^{tm1Lau}

Gene Name

nitric oxide synthase 2, inducible, macrophage

Allele Name

targeted mutation 1, Victor E Laubach

Strain Name

B6;129P2-*Nos2^{tm1Lau}/J*

Strain Former & Common Name(s): B6;129P-*Nos2^{tm1Lau}*; B6;129P2-*Nos2^{tm1Lau}*

Stock Number

002596

Strain Name

B6.129P2-*Nos2^{tm1Lau}/J*

Strain Former & Common Name(s): B6.129P2-*Nos2^{tm1Lau}*; C57BL/6-*Nos2^{tm1Lau}*

Stock Number

002609

Application(s)

Inflammation (Inflammatory bowel disease)

Additional Research Areas

Internal/Organ Research

Phenotype

Mice homozygous for the *Nos2^{tm1Lau}* targeted mutation resemble wildtype mice in appearance and histology. Homozygotes are viable and fertile. Unlike *Nos1* and *Nos3*, *Nos2* is synthesized *de novo* in response to a variety of inflammatory stimuli. Induction of *Nos2* results in the production of large amounts of nitric oxide (NO) over prolonged periods of time. Excessive NO production has been shown to be beneficial through its antitumor and antimicrobial activities. It is also thought to cause tissue damage and contribute to pathology in a variety of inflammatory conditions including rheumatoid arthritis, inflammatory bowel disease, cardiac allograft rejection, hepatotoxicity, myocardial ischemia-reperfusion and septic shock. NO has been demonstrated to play a role in the regulation of blood pressure and hemodynamics. In an LPS-induced model of septic shock, *Nos2^{tm1Lau}* homozygotes had virtually no serum NO response, but were susceptible to LPS-induced death. *Nos2^{tm1Lau}* homozygotes exhibit altered responses to *M. bovis* (BCG), systemic *E. coli* infection, *M. tuberculosis* and *M. pulmonis*. In addition, wound

Primary Reference healing properties of fibroblasts are impaired in *Nos2^{tm1Lau}* homozygotes. Also known as iNOS. Laubach VE, Shesely EG, Smithies O, Sherman PA. 1995. Mice lacking inducible nitric oxide synthase are not resistant to lipopolysaccharide-induced death. *Proc Natl Acad Sci USA* 92:10688-92.

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|--|---|
| Symbol <i>Tbx21^{tm1Glm}</i> | Gene Name T-box 21 |
| | Allele Name targeted mutation 1, Laurie H Glimcher |
| Strain Name | B6.129S6-<i>Tbx21^{tm1Glm}</i>/J |
| Stock Number | 004648 |
| General Terms & Conditions | MICE are not available for supply to companies or for profit entities. www.jax.org/jaxmice/orders/legalletter/004432_harvardlegalletter |
| Application(s) | Immunodeficiency (Asthma); Inflammation (Asthma) (Inflammatory bowel disease) |
| Phenotype | Mice that are homozygous for the targeted mutation are viable, fertile and normal in size. No gene product (mRNA or protein) is detected in isolated lymph node T cells by Northern or Western blot analysis. T cells from the homozygotes do not produce the TH1-type cytokine interferon gamma and secrete elevated levels of TH2-type cytokines in response to <i>in vitro</i> T cell receptor (TCR) cross-linking and <i>in vivo</i> protein antigen immunization. Additionally, mice homozygous for the targeted mutation on this genetic background are susceptible to <i>Leishmania major</i> infections. Without induced sensitization or challenge, female homozygotes display hyper-responsiveness (AHR) with resulting airway remodeling similar to characteristics of asthma. Histological analysis of lung tissue from female homozygous mice, aged 4 to 6 weeks, reveals eosinophil and lymphocyte infiltration of peribronchial and perivenular tissue, thickening of the subepithelial collagen layer, and increased numbers of myofibroblast cells in bronchial tissue. Bronchial alveolar lavage fluid contains elevated levels of TGFB1 (TGF-beta 1), TNF (TNF-alpha), IL4 and IL13. Mice heterozygous for the targeted mutation display an intermediate phenotype. This mutant mouse strain represents a model that may be useful in studies of acute and chronic human asthma and chronic intestinal inflammation. |
| Primary Reference | Finotto S, Neurath MF, Glickman JN, Qin S, Lehr HA, Green FH, Ackerman K, Haley K, Galle PR, Szabo SJ, Drazen JM, De Sanctis GT, Glimcher LH. 2002. Development of spontaneous airway changes consistent with human asthma in mice lacking T-bet. <i>Science</i> 295:336-8. |

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|---|--|
| Symbol <i>Tcra^{tm1Mom}</i> | Gene Name T-cell receptor alpha chain |
| | Allele Name targeted mutation 1, Peter Mombaerts |
| Strain Name | B6;129S2-<i>Tcra^{tm1Mom}</i>/J |
| Stock Number | 002115 |
| Strain Name | B6.129S2-<i>Tcra^{tm1Mom}</i>/J |
| Stock Number | 002116 |
| General Terms & Conditions | For profit entities require a license from MIT prior to shipping. www.jax.org/jaxmice/orders/legalletter/mitlegalletter |
| Application(s) | CD Antigens, Antigen Receptors, and Histocompatibility Markers; Immunodeficiency; Inflammation (Inflammatory bowel disease); T Cell Receptor Signaling Defects |
| Additional Research Areas | Hematological Research; Research Tools |
| Phenotype | Mice homozygous mice for the <i>Tcra^{tm1Mom}</i> targeted mutation are viable and fertile. They are deficient in the alpha beta T-cell receptor. The thymus is devoid of CD4 ⁺ CD8 ⁻ and CD4 ⁻ CD8 ⁺ cells. Normal numbers of CD4 ⁺ CD8 ⁺ cells are retained without the IL2 receptor. There are normal numbers of CD4 ⁻ CD8 ⁺ cells. ~1% of the thymocytes express the gamma delta TCR. Mice may develop inflammatory bowel disease beginning at ~4-6 months of age. |

Primary Reference Mombaerts P, Clarke AR, Rudnicki MA, Iacomini J, Itohara S, Lafaille JJ, Wang L, Ichikawa Y, Jaenisch R, Hooper ML, et al. 1992. Mutations in T-cell antigen receptor genes alpha and beta block thymocyte development at different stages. published erratum appears in *Nature* 1992 Dec 3;360(6403):491] *Nature* 360:225-31.

Symbol
Tcra^{tm1Mom}
Tcrb^{tm1Mom}

Gene Name **T-cell receptor alpha chain; T-cell receptor beta chain**
Allele Name **targeted mutation 1, Peter Mombaerts**

Strain Name **C.Cg-*Tcra^{tm1Mom} Tcrb^{tm1Mom}/J***

Strain Former & Common Name(s): C.Cg-*Tcra^{tm1Mom} Tcrb^{tm1Mom}*

Stock Number **004364**

Application(s) CD Antigens, Antigen Receptors, and Histocompatibility Markers; Immunodeficiency; Inflammation (Inflammatory bowel disease); T Cell Receptor Signaling Defects

Additional Research Areas Hematological Research; Research Tools

Phenotype Mice that are homozygous for the targeted mutation are viable and fertile. Mutant mice display early stage arrest of alpha beta thymocyte differentiation. Mice may develop inflammatory bowel disease.

Primary Reference Curotto de Lafaille MA, Muriglan S, Sunshine MJ, Lei Y, Kutchukhidze N, Furtado GC, Wensky AK, Olivares-Villagomez D, Lafaille JJ. 2001. Hyper immunoglobulin E response in mice with monoclonal populations of B and T lymphocytes. *J Exp Med* 194:1349-59.

Symbol
Tcrb^{tm1Mom}

Gene Name **T-cell receptor beta chain**
Allele Name **targeted mutation 1, Peter Mombaerts**

Strain Name **B6;129P2-*Tcrb^{tm1Mom}/J***

Strain Former & Common Name(s): B6;129P-*Tcrb^{tm1Mom}*; B6;129P2-*Tcrb^{tm1Mom}*

Stock Number **002117**

Strain Name **B6.129P2-*Tcrb^{tm1Mom}/J***

Strain Former & Common Name(s): B6.129P2-*Tcrb^{tm1Mom}*; C57BL/6J-*Tcrb^{tm1Mom}*

Stock Number **002118**

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Application(s) CD Antigens, Antigen Receptors, and Histocompatibility Markers; Immunodeficiency; Inflammation (Inflammatory bowel disease); T Cell Receptor Signaling Defects; Research Tools

Additional Research Areas Hematological Research

Phenotype Mice homozygous for the *Tcrb^{tm1Mom}* targeted mutation are viable and fertile. Mice are deficient in alpha beta T-cell receptor. The total number of cells in the thymus is ~8% that of wildtype; CD4⁺CD8⁺ cells ~6% of wildtype. The proportion of CD4⁺CD8⁻ (IL2 receptor positive) cells increases to about 50% of the total cell number. Alpha beta thymocyte differentiation is blocked at an earlier stage than the *Tcra^{tm1Mom}* strain. There is normal differentiation of gamma delta thymocytes. Mice may develop inflammatory bowel disease beginning at ~4-6 months of age.

Primary Reference Mombaerts P, Clarke AR, Rudnicki MA, Iacomini J, Itohara S, Lafaille JJ, Wang L, Ichikawa Y, Jaenisch R, Hooper ML, et al. 1992. Mutations in T-cell antigen receptor genes alpha and beta block thymocyte development at different stages. published erratum appears in *Nature* 1992 Dec 3;360(6403):491] *Nature* 360:225-31.

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|--|---|
| Symbol <i>Tcrb^{tm1Mom}</i> <i>Tcrd^{tm1Mom}</i> | Gene Name T-cell receptor beta chain; T-cell receptor delta chain Allele Name targeted mutation 1, Peter Mombaerts |
| Strain Name | B6;129P-<i>Tcrb^{tm1Mom}</i> <i>Tcrd^{tm1Mom}</i>/J |
| Stock Number | Strain Former & Common Name(s): B6;129- <i>Tcrb^{tm1Mom}</i> <i>Tcrd^{tm1Mom}</i> ; B6;129P- <i>Tcrb^{tm1Mom}</i> <i>Tcrd^{tm1Mom}</i> 002121 |
| Strain Name | B6.129P2-<i>Tcrb^{tm1Mom}</i> <i>Tcrd^{tm1Mom}</i>/J |
| Stock Number | Strain Former & Common Name(s): B6.129P2- <i>Tcrb^{tm1Mom}</i> <i>Tcrd^{tm1Mom}</i> ; C57BL/6J- <i>Tcrb^{tm1Mom}</i> <i>Tcrd^{tm1Mom}</i> 002122 |
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| Application(s) | CD Antigens, Antigen Receptors, and Histocompatibility Markers; Immunodeficiency; Inflammation (Inflammatory bowel disease); T Cell Receptor Signaling Defects; Research Tools |
| Additional Research Areas | Hematological Research |
| Phenotype | Mice homozygous for both the <i>Tcrb^{tm1Mom}</i> and the <i>Tcrd^{tm1Mom}</i> targeted mutations express no alpha beta T-cell receptor nor any gamma delta T-cell receptor. Under certain housing conditions homozygous mutant mice develop mild inflammatory bowel disease. |
| Primary Reference | Mombaerts P, Clarke AR, Rudnicki MA, Iacomini J, Itohara S, Lafaille JJ, Wang L, Ichikawa Y, Jaenisch R, Hooper ML, et al. 1992. Mutations in T-cell antigen receptor genes alpha and beta block thymocyte development at different stages. published erratum appears in <i>Nature</i> 1992 Dec 3;360(6403):491 <i>Nature</i> 360:225-31. |

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|---|---|
| Symbol <i>Tcrd^{tm1Mom}</i> | Gene Name T-cell receptor delta chain Allele Name targeted mutation 1, Peter Mombaerts |
| Strain Name | B6;129P2-<i>Tcrd^{tm1Mom}</i>/J |
| Stock Number | Strain Former & Common Name(s): B6;129P- <i>Tcrd^{tm1Mom}</i> ; B6;129P2- <i>Tcrd^{tm1Mom}</i> 002119 |
| Strain Name | B6.129P2-<i>Tcrd^{tm1Mom}</i>/J |
| Stock Number | Strain Former & Common Name(s): B6.129P2- <i>Tcrd^{tm1Mom}</i> ; C57BL/6J- <i>Tcrd^{tm1Mom}</i> 002120 |
| General Terms & Conditions | For profit entities require a license from MIT prior to shipping. www.jax.org/jaxmice/orders/legalletter/mitlegalletter |
| Application(s) | CD Antigens, Antigen Receptors, and Histocompatibility Markers; Immunodeficiency; Inflammation (Inflammatory bowel disease); T Cell Receptor Signaling Defects; Research Tools |
| Additional Research Areas | Hematological Research |
| Phenotype | Mice homozygous for the <i>Tcrd^{tm1Mom}</i> targeted mutation are viable and fertile. Gamma delta T-cell receptor expression is deficient in all adult lymphoid and epithelial organs. There is normal development of the alpha beta T-cell lineage. Patterns of CD4 ⁺ CD8 ⁻ and CD4 ⁺ CD8 ⁺ alpha beta T-cells are apparently normal. Mice do not develop inflammatory bowel disease. |
| Primary Reference | Itohara S, Mombaerts P, Lafaille J, Iacomini J, Nelson A, Clarke AR, Hooper ML, Farr A, Tonegawa S. 1993. T cell receptor delta gene mutant mice: independent generation of alpha beta T cells and programmed rearrangements of gamma delta TCR genes. <i>Cell</i> 72:337-48. |

| | |
|---------------------------------------|---|
| Symbol Was^{tm1Sbs} | Gene Name iskott-Aldrich syndrome homolog (human) |
| | Allele Name targeted mutation 1, Scott B Snapper |
| Strain Name | 129S6/SvEvTac-Was^{tm1Sbs}/J |
| Stock Number | Strain Former & Common Name(s): 129/SvEv-Wasp ^{tm1Sbs} ; 129S6/SvEvTac-Was ^{tm1Sbs} 003292 |
| Application(s) | Immunodeficiency; Inflammation (Inflammatory bowel disease); Intracellular Signaling Molecules; Lymphoid Tissue Defects; T Cell Receptor Signaling Defects |
| Additional Research Areas | Hematological Research; Internal/Organ Research; Mouse/Human Gene Homologs |
| Phenotype | WAS-deficient mice are viable and fertile. Mutant mice show normal lymphocyte development, serum immunoglobulin (Ig) levels and antibody responses. However, peripheral blood lymphocyte counts and platelet numbers are reduced in these mice. Development of chronic colitis is also observed. <i>In vitro</i> , WAS-deficient T cells show markedly impaired proliferative responses to anti-CD3e mediated stimulation. The Was gene is X-linked, so hemizygous males are WAS deficient. |
| Primary Reference | Snapper SB, Rosen FS, Mizoguchi E, Cohen P, Khan W, Liu CH, Hagemann TL, Kwan SP, Ferrini R, Davidson L, Bhan AK, Alt FW 1998. Wiskott-Aldrich syndrome protein-deficient mice reveal a role for WASP in T but not B cell activation. <i>Immunity</i> 9:81-91. |