The Jackson Laboratory
BAR HARBOR, MAINE USA

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Inside:





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NEWS FOR USERS OF JAX® MICE

# JAX | NOTES™

*Setting the Gold Standard for Genetic Purity™*

## JAX® MICE IN SPACE: A NEW ENDEAVOR

C57BL/6J mice (Stock Number 000664) from The Jackson Laboratory joined the crew of space shuttle *Endeavor* for a 12 day mission to the International Space Station (ISS). After several scrubbed launch attempts, mission STS-108 launched from the Kennedy Space Center on Wednesday, December 5th, 2001. This was the 12th space shuttle flight to the ISS.

On Earth, millions of people suffer from osteoporosis, a disease characterized by low bone mass and structural deterioration of bone tissue that leads to bone fragility. Cosmonauts and astronauts that served on the Mir space station showed significant post–mission loss of bone mass. BioServe Space Technologies, a non-profit NASA-sponsored Commercial Space Center at the University of Colorado, coordinated experiments with commercial sponsor Amgen Inc. to study the effects of microgravity on bone loss in C57BL/6J mice.

Amgen developed a drug called osteoprotegerin (OPG) that is aimed at eliminating rapid bone loss (Kostenuik *et al.*, 2001). OPG functions to prevent the formation of osteoclasts, which are multinuclear cells that resorb bone tissue. C57BL/6J mice were selected as flight mice because of their relatively low bone density (Beamer *et al.*, 1996) and wide use in biomedical research. Approximately 24 hours prior to the launch, 12 mice were given OPG and another 12 were given a placebo. Following the mission, the mice were examined to determine whether OPG abated bone loss.

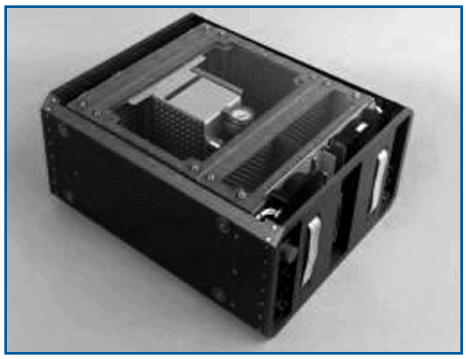

***Figure 1. C57BL/6J mice were housed in Animal Enclosure Modules (AEM) during space flight on Space Shuttle Endeavor.***

Scientists from Loma Linda University, The University of Colorado, Tulane University, and The Jackson Laboratory are collaborating with Amgen and BioServe Space Technologies to further analyze tissues from the flight mice. At The Jackson Laboratory, Wesley Beamer, Ph.D. will support Amgen's analysis of bone density by measuring microstructural variables in vertebrae using micro-CT-tomography; Luanne Peters, Ph.D. will investigate the cell membranes and cytoskeletons of erythrocytes; and David Bergstrom, Ph.D. will study otoliths, mineral concretions located adjacent to neuronal sensory cells in the inner ear.

The C57BL/6J flight mice were housed in NASA's Animal Enclosure Modules (AEM), habitats designed to house mice in a microgravity environment (Figure 1). Custom mouse diet was affixed to the walls of each AEM with an edible adhesive to maximize the availability of food. Water was dispensed from collapsible plastic bags that were surrounded and compressed by spring devices. Waste material was collected onto a filter at one end of the AEM by continuous laminar airflow.

**Continued on page 7**

*Research News*

## GENETIC BACKGROUND EFFECTS: CAN YOUR MICE SEE?

The importance of genetic background effects on an observed phenotype is frequently overlooked and often difficult to predict. Mutations present in inbred strains or hybrids and independent of the gene of interest may significantly affect the observed characteristics of a mouse. *Pde6b*$^{rd1}$, the retinal degeneration 1 mutation (allele of phosphodiesterase 6B, cGMP, rod receptor, beta polypeptide) provides an excellent example.

*Pde6b*$^{rd1}$ is present in many commonly used inbred mouse strains (Table 1) and causes blindness by wean age (Drager and Hubel, 1978, Sidman and Green, 1965). *Pde6b*$^{rd1}$ is recessively expressed (*i.e.* blindness occurs only in mice carrying two copies of the mutated gene (*Pde6b*$^{rd1}$/*Pde6b*$^{rd1}$)). F1 hybrids of sighted and blind strains (*e.g.* B6SJLF1/J, Stock Number 100012; and B6C3FeF1-*a*/*a*, Stock Number 001022) have normal vision as they carry one wildtype allele (*Pde6b*$^{rd1}$/+) (Figure 1A). However, some transgenes and spontaneous mutations are maintained on a hybrid background in a manner that results in a mixture of sighted and blind mice.

Many transgenic stains (*e.g.* B6SJL-TgN(SOD1-G93A)1Gur, Stock Number 002726) are maintained by breeding hemizygous transgene carriers (Tg/0) to B6SJLF1/J hybrids (*Pde6b*$^{rd1}$/+). Of the progeny, 50% will carry the transgene and 25% will be homozygous for the retinal degeneration 1 mutation. The transgene and *Pde6b*$^{rd1}$ will segregate independently unless the transgene has integrated into the genome in a location that is closely linked to *Pde6b*$^{rd1}$ (Figure 1B).

Likewise, many spontaneous mutations are maintained using a cross -intercross breeding scheme, resulting in a hybrid mixture that is segregating at the *Pde6b*$^{rd1}$ locus (*e.g.* B6C3Fe-*a*/*a*-*Csf1*$^{op}$, Stock Number 000231). Simply put, some mutant mice will be blind and others sighted.

If characterization of transgenic or mutant mice requires mice to see normally, it is important to genotype mice for both the presence of the transgene or mutation of interest and the absence of *Pde6b*$^{rd1}$ in the homozygous state. *Pde6b*$^{rd1}$ maps to 57 cM on Chromosome 5 (MGD, 2002). PCR-based genotyping assays for the *Pde6b*$^{rd1}$ mutation are available (Gimenez and Montoliu, 2001; Pittler and Baehr, 1991).

| Strain | Stock Number |
|---|---|
| ABJ/Le | 000276 |
| BDP/J | 000652 |
| BUB/BnJ | 000653 |
| C3H/HeJ | 000659 |
| C3H/HeOuJ | 000635 |
| C3H/HeSnJ | 000661 |
| C3HeB/FeJ | 000658 |
| CBA/J | 000656 |
| DA/HuSn | 000660 |
| FL/1Re | 000023 |
| FVB/NJ | 001800 |
| P/J | 000679 |
| PL/J | 000680 |
| SB/Le | 000269 |
| SJL/J | 000686 |
| ST/bJ | 000688 |
| SWR/J | 000689 |
| WB/ReJ *Kit*$^{W}$/+ | 000692 |
| WC/ReJ-*Kitl*$^{Sl}$/+ | 000693 |

***Table 1. Strains homozygous for the Pde6b$^{rd1}$ mutation***

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Drager UC, Hubel DH. Studies of visual function and its decay in mice with hereditary retinal degeneration. *J Comp Neurol* 1978; 180:85-114.

Gimenez E, Montoliu L. A simple polymerase chain reaction assay for genotyping the retinal degeneration mutation (*Pdeb*$^{rd1}$) in FVB/N-derived transgenic mice. *Lab Anim* 2001; 35:153-156.

Mouse Genome Database (MGD). Mouse Genome Informatics Web Site. The Jackson Laboratory. Bar Harbor, Maine. http://www.informatics.jax.org/; January 2002.

Pittler SJ, Baehr W. Identification of a nonsense mutation in the rod photoreceptor cGMP phosphodiesterase beta-subunit gene of the rd mouse. *Proc Natl Acad Sci USA* 1991; 88:8322-8326.

Sidman RL, Green MC. Retinal degeneration in the mouse. Location of the rd locus in linkage group XVII, *J Hered* 1965; 56:23-29. •

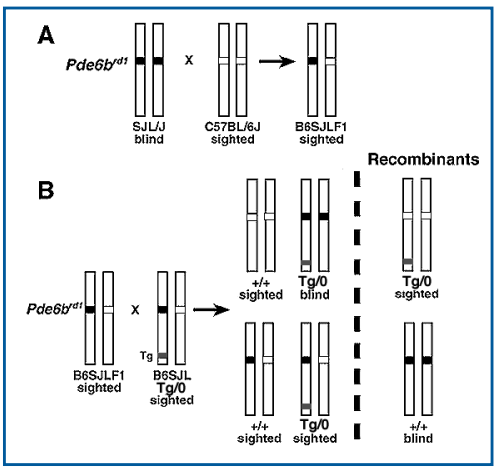


***Figure 1. Phenotypic Effects of Genes Independent of Transgene. A. SJL/J mice are homozygous for the retinal degeneration 1 mutation (gene symbol: Pde6b$^{rd1}$ ). C57BL/6J mice are wildtype at this locus. B6SJLF1 mice are sighted because Pde6b$^{rd1}$ is inherited recessively. B. Backcrossing mice hemizygous for a transgene (Tg/0) to a B6SJLF1 hybrid (Pde6b$^{rd1}$ /+) is a common breeding scheme for transgenic mice. 50% of the progeny will be carrying the transgene and 25% of the progeny will be homozygous for the retinal degeneration 1 mutation. The transgene and Pde6b$^{rd1}$ mutation will segregate independently in progeny unless the transgene has integrated into the genome in a location that is closely linked to Pde6b$^{rd1}$ mutation. Figure adapted from Linder CC. The influence of genetic background on spontaneous and genetically engineered mouse models of complex diseases. Lab Animal 2001 30:34-39, used with permission from Nature NY.***

# THE MOUSE TUMOR BIOLOGY DATABASE: INTEGRATED ACCESS TO MOUSE CANCER DATA

## http://tumor.informatics.jax.org

The Mouse Tumor Biology (MTB) Database is a public resource for cancer genetics and pathology of the mouse which is maintained at The Jackson Laboratory. This database integrates data on the frequency, incidence, genetics, and pathology of neoplastic disorders, emphasizing data on tumors that develop characteristically in different genetically defined strains of mice (inbred, mutant, and genetically engineered mice). Data are derived from literature and from research contributions. Pathological data, including images and diagnostic characteristics, are obtained from veterinary pathology laboratories.

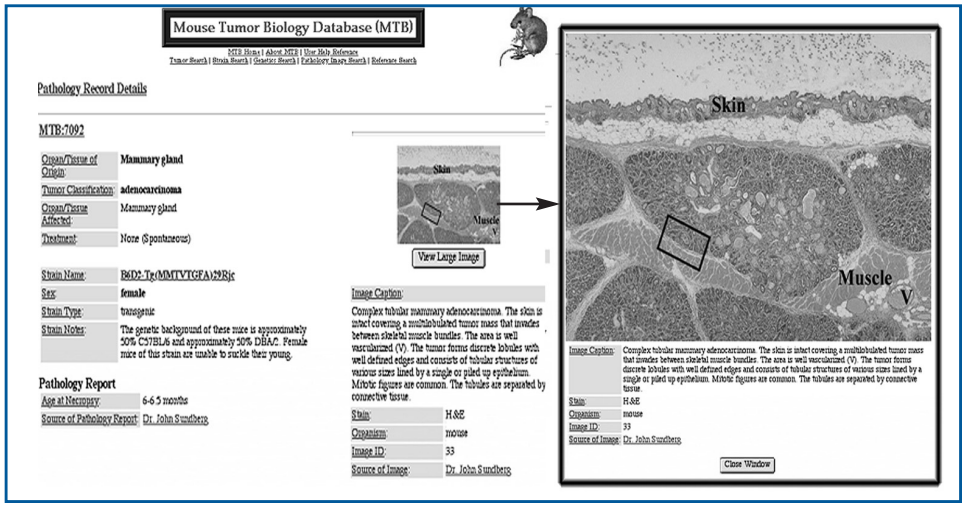


*Figure 2. Pathology Data Representation. A pathology report and captioned thumbnail image provide the detail for a pathology record. Each image has one or more enlargements available. Note the labeling of the image with terms (Muscle, Skin) and boxed area for emphasis.*

MTB provides cancer researchers with access to data on mouse models for cancer and includes information such as standardized tumor names and classifications, pathology reports, histopathological images, genomic changes in the tumor, strain genetics and names, tumor frequency and latency, and references. Relevant data in MTB are linked to related online resources such as the Mouse Genome Database (MGD), the Mouse Phenome Database (MPD), the Biology of the Mammary Gland Web Site, Festing's Listing of Inbred Strains of Mice, the JAX® Mice Web Site, and the Mouse Models of Human Cancers Consortium's (MMHCC's) Mouse Repository. MTB is designed to support the selection of strains for experimental study, the comparison of experimental models for cancer research, the evaluation of mouse genetic models of human disease, the review of patterns of mutations in specific cancers, and the identification of genes that are commonly mutated across a spectrum of cancers. By making such data readily available online, MTB provides the scientific community with a much-needed, easily accessible, central resource for rapidly finding and evaluating the ever-expanding volume of mouse tumor data.

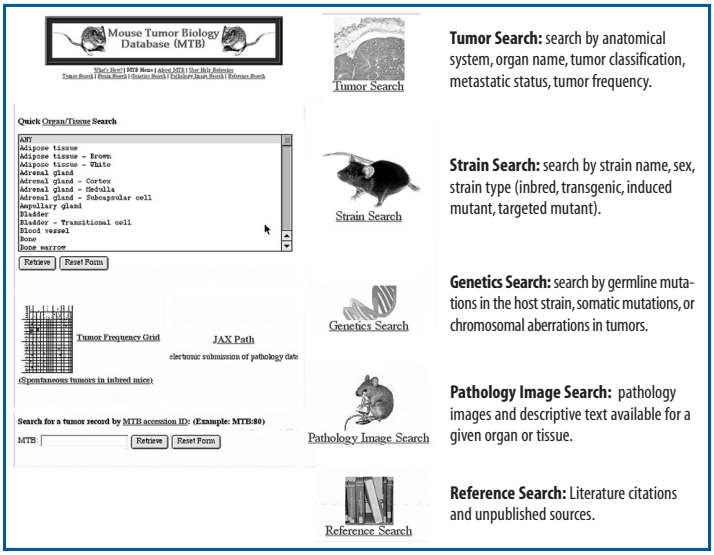


*Figure 1. Mouse Tumor Biology Database Homepage. Users can launch a general query based on organ/tissue name, access the Tumor Frequency Grid graphical query tool, or select one of the advanced query forms.*

Figures 1 and 2 illustrate some of the features of MTB. For further information, consult the "About Page" on the MTB Web site or the article cited.

MTB is supported by NCI grant CA89713.

## REFERENCES:

*(Authors in bold are Jackson Laboratory scientists)*

**Näf D, Krupke DM, Sundberg JP, Eppig JT, Bult CJ.** The Mouse Tumor Biology Database: A Public Resource for Cancer Genetics and Pathology of the Mouse. *Cancer Research* 2002; 62 (in press). •

## JAX® MICE WEB SITE HIGHLIGHTS www.jax.org/jaxmice

new **JAX® Mice News E-mail Newsletter:** "JAX® Mice News" is an e-mail newsletter for biomedical researchers that provides news related to JAX® Mice. This newsletter contains information on new mouse models available from The Jackson Laboratory, strains under development, research applications for JAX® Mice, upcoming scientific meetings, and much more. Subscribers also receive "JAX® Mice News Alerts" which are timely news bulletins relevant to expressed research interests. An on-line form is now available for requesting literature and e-mail publications at: www.jax.org/jaxmice/subscribe.

**International JAX® Mice Price List, Effective January 1, 2002:** This new price list can be accessed on-line in three easy steps: 1. Go to www.jax.org/jaxmice and select Price List & Product Guide from the Main Menu; 2. Select Download PDF File from the submenu of the Main Menu; 3. Select JAX® Mice International Price List from the text in the center of the page.

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## SELECTED PUBLICATIONS AUTHORED BY JACKSON LABORATORY SCIENTISTS

*(Authors in bold are Jackson Laboratory scientists)*

### AGING RESEARCH

**Chen J, Flurkey K, Harrison DE.** A reduced peripheral blood $CD4^+$ lymphocyte proportion is a consistent ageing phenotype. *Mech Ageing Dev* 2002; 123:145-153.

**Flurkey K,** Papaconstantinou J, **Harrison DE.** The Snell dwarf mutation $Pit1^{dw}$ can increase life span in mice. *Mech Ageing Dev* 2002; 123:121-130.

**Klebanov S, Flurkey K, Roderick TH, Archer JR, Astle MC, Chen J, Harrison DE.** Heritability of life span in mice and its implication for direct and indirect selection for longevity. *Genetica* 2000; 110:209-218.

### BIOINFORMATICS

**Blake JA, Richardson JE, Bult CJ, Kadin JA, Eppig JT and The Mouse Genome Database Group.** The Mouse Genome Database (MGD): the model organism database for the laboratory mouse. *Nucl Acids Res* 2002; 30:113-115.

### CANCER RESEARCH

**Barak Y,** Liao D, He W, Ong ES, Nelson MC, Olefsky JM, Boland R, Evans RM. Effects of peroxisome proliferator-activated receptor delta on placentation, adiposity, and colorectal cancer. *Proc Natl Acad Sci USA* 2002; 99:303-308.

### DEVELOPMENTAL BIOLOGY RESEARCH

**Albrecht KH, Eicher EM.** Evidence that *Sry* is expressed in pre-Sertoli cells and Sertoli and granulosa cells have a common precursor. *Dev Biol* 2001; 240:92-107.

### DIABETES RESEARCH

**Savinov AY,** Wong FS, **Chervonsky AV.** IFN-gamma affects homing of diabetogenic T cells. *J Immunol* 2001; 167: 6637-6643.

### GENETIC ENGINEERING

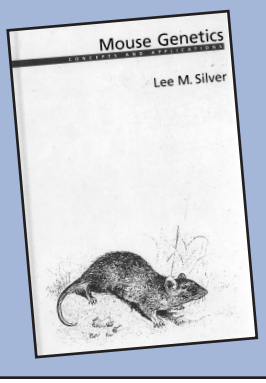
Wurst W, **Gossler A.** Gene trap strategies in ES cells. IN: Joyner AL, ed. *Gene Targeting; a Practical Approach,* 2nd ed. New York; Oxford U Press, 2000; 207-254.

### OSTEOPOROSIS RESEARCH

**Rosen CJ, Beamer WG, Donahue LR.** Defining the genetics of osteoporosis: using the mouse to understand man. *Osteoporosis Int* 2001; 12:803-810.

### REPRODUCTIVE BIOLOGY RESEARCH

**Eppig JJ.** Oocyte control of ovarian follicular development and function in mammals. *Reproduction* 2001; 122:829-838. •




The Mouse Genome Informatics (MGI) group at The Jackson Laboratory has created an electronic version of Lee Silver's *Mouse Genetics*. This book is a valuable resource for all researchers in mouse genetics. The electronic version has been enhanced by links from the text to dynamic databases at MGI and NCBI. All genes are linked to the corresponding MGI marker detail page, and most references link to Medline. All of the Tables and Figures present in the original book are available online. The book is accessible at: www.informatics.jax.org/silver/ •

*Research News*

## GENES IMPLICATED IN A MOUSE MODEL FOR PIGMENTARY GLAUCOMA

Pigmentary glaucoma is a leading cause of blindness in humans. In this disease, pigment and/or cellular debris released from the iris becomes lodged in the microvessels that drain the eye, leading to an increase in intraocular pressure, atrophy of the optic nerve, and ultimately, blindness. DBA/2J mice (Stock Number 000671) develop a disease that is strikingly similar to human pigmentary glaucoma. The disease in these mice is characterized by two genetically separable components: iris pigment dispersion (IPD) and iris stromal atrophy (ISA).

The primary characteristics of IPD include deterioration of the posterior iris pigment epithelium and dispersion of pigment. Similarly, ISA is characterized by deterioration of the anterior iris stroma, loss of stromal complexity, and the accumulation of pigment and cellular debris in the ocular drainage system.

Recently, researchers in Simon John's[1] group at The Jackson Laboratory, with collaborators from the Massachusetts Eye and Ear Infirmary, have identified the genes responsible for the IPD and ISA components of pigmentary glaucoma in DBA/2J mice (Anderson *et al.* 2002).

Using high resolution genetic mapping, John's group identified several candidate genes in a region of mouse Chromosome 6 which correlated with the IPD phenotype. Among these genes, glycoprotein (transmembrane) nmb, *Gpnmb*, was known to be expressed within pigment cells in the eye. Sequencing of *Gpnmb* from DBA/2J mice revealed a mutation creating a premature stop codon at arginine 150 of the *Gpnmb* coding sequence. This mutation results in the expression of a truncated protein that lacks several structural domains, including a putative melanocyte sorting motif and a PKD (polycystic kidney disease) domain that could mediate protein-protein interactions. The role of *Gpnmb* in IPD was confirmed by the serendipitous discovery of several DBA/2J sublines that carry wild-type alleles of *Gpnmb*. These mice had been separated from the parental DBA/2J strain and cryopreserved at The Jackson Laboratory several years ago. Studies of aged-matched wildtype, heterozygous, and homozygous mutant *Gpnmb* mice, showed that only mice homozygous for the *Gpnmb* mutant allele developed IPD, proving, unequivocally, that the *Gpnmb* mutant allele causes IPD.

Previous work conducted in the John lab suggested a role for the coat color gene *Tyrp1* in the ISA component of the pigmentary glaucoma in DBA/2J mice (Chang *et al.* 1999). DBA/2J mice are homozygous for a mutant *Tyrp1* allele ($Tyrp1^b$) that gives these mice a brown coat.

Combined with experiments from the previous study, a total of 1522 meioses from crosses segregating for *Tyrp1* and $Tyrp1^b$ have now been analyzed, and an absolute correlation between the ISA phenotype and $Tyrp1^b$ allele has been firmly established. Further, experiments in the present study show that a transgene carrying a wildtype copy of *Tyrp1* can rescue completely, the ISA phenotype in DBA/2J inbred mice. These results confirm the role of *Tyrp1* in ISA.

Having established the importance of *Gpnmb* and *Tyrp1* in IPD and ISA, respectively, Dr. John's goal now is to understand the mechanisms through which mutations in the two genes result in glaucomas. The proteins encoded by both *Gpnmb* and *Tyrp1* contain structural motifs common to melanosomal proteins, including tyrosinase, which in humans have been shown to be important in organizing and maintaining the melanogenic protein complex and melanosomal structure. John and his colleagues speculate that *Gpnmb* and *Tyrp1* have similar functions in the stability and synthesis of melanosomes, such that the mutations in these genes result in the release of

***DBA/2J mice develop a disease that is strikingly similar to human pigmentary glaucoma.***

cytotoxic intermediates in eye pigment synthesis into the eye, leading, ultimately, to glaucoma. In support of this hypothesis, the investigators also report that mice with reduced levels of eye pigment production do not show signs of glaucoma even when homozygous for the DBA/2J *Gpnmb* and *Tyrp1* mutations. Thus, induction of IPD and ISA in mice appears to depend on active pigment production.

The mechanisms underlying human glaucoma remain largely unknown. The identification of mutations in *Gpnmb* and *Tyrp1* as the causes of pigmentary glaucoma in DBA/2J mice mark an important step in unraveling the mysteries of these mechanisms, and promise new insights into the development of potential therapies for treating this disease in man.

[1] Simon John is a Howard Hughes Medical Institute Investigator.

### REFERENCES:

***(Authors in bold are Jackson Laboratory scientists)***

**Anderson MG, Smith RS, Hawes NL, Zabaleta A, Chang B, Wiggs JL, John SWM.** Mutations in genes encoding melanosomal proteins cause pigmentary glaucoma in DBA/2J mice. *Nat Genet* 2002; 30:81-85.

**Chang B, Smith RS, Hawes NL, Anderson MG, Zabaleta A, Savinova O, Roderick TH, Heckenlively JR, Davisson MT, John SWM.** Interacting loci cause severe iris atrophy and glaucoma in DBA/2J mice. *Nat Genet* 1999; 21: 405-408. •

# JAX® GEMM® Strains

*Genetically Engineered and Mutant Mice*

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**B6;129-*Cd44*$^{tm1Hbg}$**
**Stock Number:** 003899
**Applications:** Studies in immunology and inflammation research and cell biology research: cell motility and cell adhesion defects.

**129/Sv-*Cdkn1b*$^{tm1Mlf}$**
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**Applications:** Studies in cancer research, reproductive biology research (fertility defects in females), and cell biology research.

**129S4/SvJaeSor-*Gt(ROSA)26Sor*$^{tm1(FLP1)Dym}$**
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**B6.129S7-*Ifngr*$^{tm1Agt}$**
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## Expanded Ground Transportation Routes for JAX® Mice

The Jackson Laboratory is pleased to announce the expansion of our existing dedicated ground transportation routes. We have incorporated an additional 21 states and 2 provinces, including Washington, Texas, Florida, British Columbia and points in-between. This expansion will enable us to offer safe, cost effective, weekly dedicated ground transportation to over 90% of our USA and Canadian customers.

We are very excited about this enhancement. Our primary concerns are for the health and welfare of our mice, and for customer satisfaction. A comprehensive study of transit data indicates that mice delivered by dedicated truck are exposed to less stress and arrive in better condition than those traveling by air. Our goal is to control the environment of the mice from our door to yours.

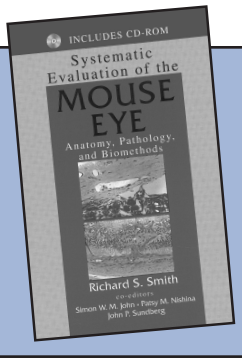
The dedicated vehicles used for this service have been modified to maintain sanitary conditions and facilitate air flow. They are equipped with environmental control systems that alert the drivers in case of malfunction. Futhermore, we have immediate access to replacement equipment should a malfunction occur en route.

We have tested and selected an alternative food and water source for use during transit. In place of our traditional canned shipping diet, the mice are packed with gel water packets and with the same pelleted diet they are fed while in our production facility. We have successfully used this food and water combination for two years in shipments to West Coast, USA and international destinations. JAX® Mice shipped by this method arrive in excellent health after journeys of up to 10 days; in fact, some have actually gained weight while in transit.

We highly recommend the use of ground transportation to help ensure the health, welfare and safety of JAX® Mice, as we are best able to control their handling and environment using this mode of transit.

Ground deliveries are made once per week. New routes are being launched in stages this year (by territory) throughout February, March, and April. To take full advantage of the new routes, existing shipments are being converted to ground transportation whenever possible. Detailed information on the new service has been forwarded to purchasing agents. If you have questions or comments, please feel free to contact Laura Mathews, Transportation Coordinator, at 800.422.6423 or 207.288.5845. We look forward to offering this improved animal transportation service to our customers. •






## New Reference Book on the Mouse Eye Edited by Jackson Laboratory Scientists

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*Questions and Answers*

## Frequently Asked Questions About Jax® Mice

**Q** ***Does the Jackson Laboratory accept new strains of mice from the scientific community?***

**A** The Jackson Laboratory accepts both genetically and non-genetically engineered mice from the research community for distribution. Researchers may submit genetically engineered strains to The Jackson Laboratory Induced Mutant Resource (IMR) for consideration. The IMR has an in-house committee, the Genetic Resources Committee, that meets monthly to discuss the strains submitted. Due to the large number of mutants being created by the scientific community, the submission process has become somewhat competitive. Our resources permit us to accept approximately 65 strains a year. The URL to our on-line submission form is: www.jax.org/resources/documents/imr/form.html

Researchers may submit new strain applications for non-genetically engineered mice directly to the Genetic Resources Committee. To access the submission form, go to www.jax.org/resources/documents/grc/grchomeout.html

**Q** ***Is there a resource available to access approved mouse gene symbols and names?***

**A** The Mouse Genome Database (MGD) contains information on mouse genetic markers, as well as other types of molecular and phenotypic data on mice. MGD is updated daily. To search for current gene symbols and names in MGD, go to www.informatics.jax.org

Two basic types of searches for gene symbols and names can be performed:

To execute a Quick Gene search, enter a single gene/QTL symbol or name in the search field and click the GO button. The search checks current names and symbols, withdrawn symbols and other names.

To use the Genes, Markers, and Phenotypes Query Form follow the links from the MGD homepage, or go directly to: www.informatics.jax.org/searches/marker_form.shtml •

## Jax® Mice in Space (Continued)

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Kostenuik PJ, Shalhoub V. Osteoprotegerin: a physiological and pharmacological inhibitor of bone resorption. *Curr Pharm Des* 2001; 7:613-35. •

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**AALAS National Meeting**
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**Society for Neuroscience Annual Meeting**
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