

# SJL

## SJL/JCrHsd

The SJL mouse is a Swiss mouse, inbred by James Lambert in 1955 from Swiss-Webster stock from three different sources that were brought to the Jackson Laboratory between 1938 and 1943. Pen-bred until 1955, when sib-mating was started.

Derived from a nucleus colony which originated at the Jackson Laboratories, Bar Harbor, Maine.

### RESEARCH APPLICATIONS

Hodgkin disease, aggression, reticulum cell sarcomas, amyloidosis, immunology.

### CHARACTERISTICS

Although the strain has been developed relatively recently, it has rapidly become widely used owing to the high incidence of reticulum cell sarcomas resembling Hodgkin's disease.

#### Animal model

The SJL mouse is an animal model for Hodgkin's disease (Kumar, 1983).

#### Anatomy

Low brain weight (Storer, 1967). Low brain weight, small spinal cord (Roderick *et al*, 1973). Cerebellum has no intraculminate fissure between vermician lobule IV and vermician lobule V (the ventral and dorsal lobules of the culmen) (contrast DBA/2) (Cooper *et al*, 1991). Low percent carcass lipid on a high-fat diet (West *et al*, 1992). Low retinal ganglion cell number (Williams *et al*, 1996). High bone density of femur (Beamer *et al*, 1996). Correlation between the organisation of the hippocampus and behavior in nine strains and three F1 hybrids (Roullet *et al*, 1990).

#### Behavior

High spontaneous fighting (Page and Glenner, 1972). Severe fighting among males housed together, beginning at about eight weeks. Most males will be killed by four-five months unless caged separately (Crispens, 1973). Very sensitive

to the induction of ataxia by diazepam. A comparative study of the sensitivity to different effects by diazepam (Crabbe *et al*, 1998).

#### Drugs

Resistant to skin ulceration by DMBA (Thomas *et al*, 1973). Resistant to induction of subcutaneous tumors by 3-methylcholanthrene (Kouri *et al*, 1973; Whitmire *et al*, 1971).

Urethra only slightly leukemogenic, but 7, 12-dimethylbenzanthrene increased lymphocytic neoplasms from 2% to 83% in young mice (East, 1970; Haran-Goeha *et al*, 1967). Resistant to X-irradiation (Roderick, 1963). Poor ovulatory response to three I.U. and seven I.U. of PMS, but response facilitated by exposure to males at latter dose rate only (Zarrow *et al*, 1971). Resistant to hyperbaric oxygen (Hill *et al*, 1968) Short survival in 90% oxygen (Lieberman and Kellog, 1967). Resistant to X-irradiation as judged by the LD<sub>50</sub> (Yuhas and Storer, 1969). Susceptible to induction of splenic amyloidosis by injection of casein (Clerici, 1972). Susceptible to induction of lymphoid and myeloid leukemia by DMBA (Crispens, 1973). Resistant to biliary tract injury following oral dosing with 500 micrograms of the fungal toxin sporidesmin (Bhathal *et al*, 1990). Airways hyporeactive to acetylcholine (Zhang *et al*, 1995). Susceptible to ozone-induced decreases of tracheal potential (Takahashi *et al*, 1995). Low voluntary consumption of morphine in two-bottle choice situation (Belknap *et al*, 1993). Susceptible to weight loss induced by cocaine, but this is

attenuated by anisomycin (cf C3H, CBA) (Shimosato *et al*, 1994).

### Genetics

#### Coat color genes

- *A, B, c, D, p*: albino.

#### Histocompatibility

- *H-2<sup>s</sup>*.

#### Biochemical markers

- *Apoa-1<sup>a</sup>, Car-2<sup>b</sup>, Es-1<sup>b</sup>, Es-2<sup>b</sup>, Gpi-1<sup>a</sup>, Hbb<sup>s</sup>, Pep-3<sup>b</sup>, Pgm-1<sup>b</sup>, Pgm-2<sup>a</sup>, Trf<sup>b</sup>*.

Carries the pink-eyed dilution gene, *p*, which is derived from Asian mice of the *Mus musculus* type (Brilliant *et al*, 1994). This strain carries the *Mus musculus domesticus* Y-chromosome, while others have the *M. m. musculus* type (Nishioka, 1987).





## Immunology

Susceptible to induction of experimental allergic encephalomyelitis (EAE) (Levine and Sowinski, 1973; Goverman and Brabb, 1996). High susceptibility to induction of EAE, but moderate mortality (Lindsey, 1996).

The induction and effector phases of acute EAE are apparently controlled by the combination of H-2 and HSF genes. A combination of the correct H-2 haplotype and histamine sensitivity is required for the development of acute EAE (Linthicum and Frelinger, 1986). Pretreatment with whole body ultraviolet irradiation, a process that selectively interferes with antigen-presenting cell function in other systems, protected animals from paralytic signs and pathological manifestations in acute EAE (Hauser *et al*, 1984). EAE can be modulated with anti-T suppressor factor antibodies (Perry *et al*, 1988). Low lymphocyte phytohemagglutinin response (Heiniger *et al*, 1975). Poor immune response to small doses of bovine gamma-globulin (Levine and Vaz, 1970). Poor immune response to DNP-keyhole-limpet hemocyanin (Borel and Kilham, 1974). No immune response to GAT (random terpolymer of Glu<sup>60</sup>, Ala<sup>30</sup>, Tyr<sup>10</sup>) (Dorf *et al*, 1974). Discriminator between 'H' and 'L' sheep erythrocytes (McCarthy and Dutton, 1975). Very sensitive to anaphylactic shock (Treadwell, 1969). Resistant to induction of immunological tolerance (Fujiwara and Cinader, 1974). Poor immune response to (Pro<sup>66</sup>, Gly<sup>34</sup>)<sub>n</sub> (Fuchs *et al*, 1974). High susceptibility to IgE- and IgG1-mediated passive cutaneous anaphylaxis (De Souza *et al*, 1974). Erythrocytes have a high agglutinability (Rubinstein *et al*, 1974). Low response to Dextran (Blomberg *et al*, 1972).

Immune response to type-III pneumococcal polysaccharide declines by 42 weeks, in contrast to BALB/c and C3H (Smith, 1976). Susceptible to induction of experimental autoimmune thyroiditis (Vladutiu and Rose, 1971). Thymocytes exhibit a periodicity (5-9 days) in their response to hormonal stimulation with isoproterenol. This is expressed in large changes in the intensity of the response (peak levels of intracellular cAMP which vary approximately 6-fold), and in the response pattern, i.e., in the occurrence or non-occurrence of an immediate hormone-induced desensitisation. In contrast, C57BL/6 thymocytes have a homogeneous response pattern (Riven-Kreitman *et al*, 1990). Resistant to immunosuppression of contact hypersensitivity by ultraviolet B light (Noonan and Hoffman, 1994). Low natural killer cell response to the immunostimulant 7-allyl-8-oxoguanosine (Pope *et al*, 1994). Has defective T cell receptor-induced interleukin-4 production and absence of T-cells with the NK1.1 antigen. However, natural-killer-like T-cells develop normally in spite of these defects (Beutner *et al*, 1997). Mast cells grow faster in culture and have more than twice the amount of histamine and TNF-alpha in their granules than BALB/c (Bebo *et al*, 1996). High level of serum complement C5 (Lynch and Kay, 1995). In SJL, NK1.1+ T cells, a specialized set of T cells that recognize CD1, accumulate in the liver at ageing. (Murakami *et al*, 1998). Sensitive to the induction of lupus autoimmunity by peptide immunisation in contrast with other strains (James and Harley, 1998)

## Infection

Encephalomyocarditis virus causes diabetes mellitus (Boucher *et al*, 1975). Susceptible to Friend S and B virus (Klein, 1975). High susceptibility to develop leukemia on infection with Friend virus (Dietz and Rich, 1972). Sensitive to cytomegalovirus (Price *et al*, 1990). Resistant to measles virus (Rager-Zisman *et al*, 1976; Neighbour *et al*, 1978).

Develop flaccid paralysis and survivors develop a distinct neurological disorder associated with marked mononuclear cell infiltration and active demyelination in spinal cord after intracerebral inoculation with Theiler's encephalomyelitis virus. Incubation period may be 2-3 months (Lipton and Dal Canto, 1976). Resistant to street rabies virus (SRV) injected via the intraperitoneal route (Perry and Lodmell 1991). Develops herpes simplex encephalitis (HSE) resembling the human condition, following intranasal infection with a neurovirulent clinical isolate of herpes simplex virus type 1 (contrast 9 other strains) (Hudson *et al*, 1991). Resistant to carditis on infection with Lyme borreliosis (*Borrelia burgdorferi*) (contrast C3H, SWR, BALB/c) (Barthold *et al*, 1990). High eosinophilia on infection with the helminth *Mesocestoides corti* and highly susceptible to infection with the parasite. Larval burdens at 21 days after infection with 100 tetrathyridia being considerably higher (greater than 1000) than all other strains except NIH, which was comparable. (Lammas *et al*, 1990). Susceptible to infection by *Helicobacter felis* with moderate to severe chronic active gastritis in the body of the stomach, which increased over time (Sakagami *et al*, 1996).



### Life-span and spontaneous disease

Short life-span in conventional conditions (8/22 = 472 days in males, 3/22 = 395 days in females). High gross tumor incidence (Storer, 1966). Reticulum cell sarcomas appear in about 90% of animals at an average age of about 13 months (Murphy, 1963; Crispens, 1973; Fujinaga *et al*, 1970). These first appear in the Peyer's patches and mesenteric lymph nodes and later in the spleen, liver, thymus and other lymph nodes (Crispens, 1973). Most of the tumors are pleomorphic or mixed-cell types commonly called type-B reticulum cell neoplasms by Dunn, but a few are type-A histiocytomas. The unusual feature of the SJL reticulum cell tumors is their regular and early appearance, with the preneoplastic lesion detectable as early as 22 days (Potter, 1972). Tumor development as well as autoimmunity may result from an effective amplification of the immune response (Owens and Bonavida, 1976). Leukemia 83% (Myers *et al*, 1970). High incidence of spontaneous amyloidosis, possibly associated with fighting (Page and Glenner, 1972).

Develops gamma-1 and gamma-2 paraproteinemia (Wanebo *et al*, 1966).

Hyperplastic neuroretinopathy and disorders of pigment epithelial cells with a high incidence of subretinal tumor is present at 9 days (Caffe *et al*, 1993). Disease patterns and life-span in ageing mice have been described by Myers (1978).

### Physiology and biochemistry

Low plasma cholesterol at 24 weeks (Weibust, 1973). High metabolic rate (Storer, 1967). Low serum ceruloplasmin levels in females but intermediate in males (Meier and MacPike, 1968). High systolic blood pressure (Schlager and Weibust, 1967). Low plasma cholinesterase activity in males (Angel *et al*, 1967). High mean heart rate, but low mean heart rate adaptation (Blizard and Welty, 1971). High brain sphingosine and low brain sterol (Sampugna *et al*, 1975). Low hepatic delta-aminolevulinic acid synthetase activity after DDC treatment (Gross and Hutton, 1971). Venous blood has a low pH (Dagg, 1966).

Resistant to the development of atherosclerosis on a semi-synthetic high fat diet (Nishina *et al*, 1993). High intrinsic myogenicity of muscle cells both in-vivo and in-vitro (Maley *et al*, 1994; Mitchell *et al*, 1995). Genetic study to compare the reaction of SJL and eight other strains to high-fat and high-cholesterol diets (Paigen, 1995)

### Miscellaneous

General biological data on the strain have been reviewed by Crispens (1973). The relationship of genotype, sex, body weight, and growth parameters to lifespan in inbred and hybrid mice is described by Ingram *et al* (1982). Characteristics of the SJL strain have been described by Festing (1997) and Lyon *et al*, (1996).

### Reproduction

When females were mated to BALB/cBm males to determine the role of the maternal genome in the sex reversing non-disjunction of the Y chromosome, SJL/J females produced 39.5-41.5% males and 2.4-2.8% hermaphrodites (Whitten *et al*, 1991).



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