

BALB/C (Bagg's Albino)

BALB/cOlaHsd

Albino stock acquired by H. Bagg in 1913 and therefore called "Bagg albino" or BALB. In 1923, inbred by MacDowell, Cold Spring Harbor, NY, USA. In 1932, at F26 to Snell, who added the 'c' for albino.

Obtained by Laboratory Animals Centre, Carshalton from the Jackson Laboratory, Bar Harbor, ME, USA in 1955. To Clinical Research Centre, Harrow, then to Olac in 1976, to Harlan through acquisition of Olac. Harlan became Envigo in 2015, then Envigo was acquired by Inotiv in 2021. Harlan Laboratories Israel became an independent company in 2023.

RESEARCH APPLICATIONS

Immunology, plasmacytomas, monoclonal antibodies, behavior, aggression, low mammary tumor incidence, corpus callosum, hippocampus and parasitology.

CHARACTERISTICS

The BALB/c is used as a general-purpose strain in many disciplines. Well known for the production of plasmacytomas on injection with mineral oil. These tumors form the basis of the production of monoclonal antibodies.

Anatomy

Large brain weight (Storer, 1967; Roderick et al, 1973; Wahlsten et al, 1975). Large brain to body weight ratio. Large spinal cord (Roderick et al, 1973). Large relative kidney weight (Schlager, 1968). Large forebrain and hippocampus volume (Wimer et al, 1969). Large number of A10 dopaminergic neurons in midbrain region (Bernardini et al, 1991). Corpus callosum absent in 39% of animals (Wahlsten, 1974).

This is associated with slow growth of the medial septum subadjacent to the cavum septi. (Wahlsten and Bulman- Fleming, 1994). Absence of corpus callosum related to retarded formation of the hippocampal commissure in this strain and in 129/J mice (Livy and Wahlsten, 1997). Low bone density of femur (Beamer et al, 1996). Anatomy of Ammon's horn (hippocampus and dentate gyrus) different from that of seven other strains (Barber et al, 1974).

High erythrocyte count, high hematocrit, high hemoglobin (Russell et al, 1951).

Large spleen at all ages (Albert et al, 1966). Accessory spleens in about 21% of animals, and number of nipples commonly exceeds five pairs (Hummel et

al, 1966). Occasional (less than 2%) cases of visceral inversion (Hummel and Chapman, 1959). Small pituitary (Sinha et al, 1975). Large proportion of spermhead abnormalities (44%) (Styrna et al, 1991).

Low level of spontaneous sister chromatid exchange (Nishi et al, 1993). Provides a sensitive and reproducible model of focal and global brain ischemia (Barone et al, 1993). Important blood volume: 10.35 ml/100 g (Vacha, 1975).

Behavior

High intra-strain aggression, low open- field activity, high tail rattling but low social grooming during aggressive encounters (Southwick and Clark, 1966). Low open- field activity (Thompson, 1953). High spontaneous locomotor activity (Nikulina et al, 1991). Long time of immobility in a forced swimming test (Nikulina et al, 1991). Short latency to cross barrier in maze, high urination and defecation in test apparatus (McClearn et al, 1970). Low wheel activity (Messeri et al, 1972). Low avoidance conditionability (Royce, 1972) and low shock avoidance learning in males (Royce et al, 1971). Poor shock avoidance learning (Wahlsten, 1973). Low alcohol preference ratio



(McClearn, 1965; Rodgers, 1966). High social dominance of males in competition for females (DeFries and McClearn, 1970). Highbalsa- wood gnawing activity (Fawdington and Festing, 1980). Exhibit hypersecretion of corticosterone and marked brain catecholamine alterations and disruption of Morris water maze performance following stressors such as footshock. However, performance deficits were prevented by cross fostering to C57BL/6 foster mothers (Zaharia *et al*, 1996).

Drugs

Susceptible to skin ulceration by 7,12- dimethylbenz(a) anthracene (DMBA) (Thomas *et al*, 1973). Sensitive to the development of uterine tumors following treatment with DMBA at 4- weeks of age (Tsubura *et al*, 1993). Sensitive to the induction of skin tumors by methylnitrosourea in methanol (Lijinsky *et al*, 1991). Susceptible to tumorinduction by 3-methylcholanthrene (Whitmire *et al*, 1971). High incidence of lung tumors after administration of methylcholanthrene by gavage (Akamatsu and Barton, 1974). Susceptible to induction of leukemia but resistant to induction of liver tumors by neonatally administered DMBA (Flaks, 1968). High incidence of interstitial tumors of testis induced by stilboestrol, high incidence of hemangioendotheliomas, particularly in interscapular fat pad and lung in mice treated with O-aminoazotoluene (Heston, 1963). Injection of mineral oil i.p. induces a high incidence of transplantable plasmacytomas (myelomas). Bence Jones proteins include kappa and lambda light chains and the two-chain IgA protein. 60% of tumors are of the IgA type (Potter, 1972). Susceptibility appears to be mediated by two genes on chromosome 4 (Potter *et al*, 1994). Susceptible to daunomycin-induced nephrosis (Kimura *et al*, 1993). Sensitive to Xirradiation (Roderick, 1963; Storer, 1966). Low LD50 to X-irradiation (Yuhass and

Storer, 1969). Nicotine increases shock avoidance learning (Bovet *et al*, 1966). Sensitive to insulin (Brown, 1965). Poor ovulatory response to PMS at both 3 IU and 7 IU, but response increased by exposure to males (Zarrow *et al*, 1971). Low locomotor excitation after treatment with D-amphetamine (Babbini *et al*, 1974). Resistant to hyperbaric oxygen (Hill *et al*, 1968). Insensitive (eosinophil response) to cortisone acetate (Wragg and Speirs, 1952). Low sensitivity to induction of malformed ribs and vertebrae by hypoxia on ninth day of gestation (Dagg, 1966). Sensitive to chloroform toxicity (Christensen *et al*, 1963). Resistant to toxic effects of isoniazid (Taylor, 1976b). Resistant to neurotoxic effects of monocrotophos (Rao *et al*, 1991). High transient increase in renal lipid peroxidation following treatment with nickel (Misra *et al*, 1991). Resistant to biliary tract injury following oral dosing with 500 micrograms of the fungal toxin sporidesmin, but the injury is much more persistent than in SJL and was accompanied by periductal fibrosis and occasionally by obliteration of ducts typical of sclerosing cholangitis (Bhathal *et al*, 1990). High LD50 following injection of butylated hydroxytoluene (BHT) (Kehrer and DiGiovanni 1990). High histamine release from peritoneal mast cells induced by compound 48/80, a calcium dependent histamine releaser (Toda *et al*, 1989). High histamine release from peritoneal mast cells induced by Ca²⁺ ionophore A23187 (contrast C57BL/6) (Toda *et al*, 1989). Cultured mast cells grow more slowly and release

less histamine and TNF-alpha following anti-DBN IgE antibody treatment than those of strain SJL (Bebo *et al*, 1996).

Highly sensitive to the induction of catalepsy by haloperidol associated with midbrain dopamine D2 receptor density levels (Kanes *et al*, 1993). Resistant to both acute and chronic cadmium toxicity (contrast NFS) (Abshire and Waalkes, 1994). However, cadmium can induce hematopoietic and suppress pulmonary tumors in these mice (Waalkes and Rehm, 1994). Resistant to weight loss induced by cocaine (Shimosato *et al*, 1994). Clonidine induces a strong aggressive behavioral response (Nikulina and Klimek, 1993). More resistant to acute toxic effects of aflatoxin B-1 than C57BL/6 (Almeida *et al*, 1996). The IgE response following topical application has been used to predict which chemicals may have the potential to cause sensitization of the respiratory tract (Hilton *et al*, 1996). More susceptible to the development of micronuclei than C57BL/6 or DBA/2 following treatment with clastogenic base analogues and nucleosides (Sato *et al*, 1993). Estrogen does not induce an increase in VLDL and LDL-cholesterol (like C3H contrast C57BL/6 and C57L) (Srivastava, 1995).

Genetics

Coat color genes

- A, b, c, D: albino.

Histocompatibility

- H-2^d, Thy-1^b

Biochemical markers

- ApoA-1^b, Car-2^b, Es-1^b, Es-2^b, Es-3^a, Gpd-1^b, Gpi-1^a, Hba^b, Hbb^d, Idh-1^a, Ldr-1^a, Mod-1^a, Mup-1^a, Pep-3^a, Pgm-1^a, Pgm-2^a, Trf^b.

1991). Low immunological response to *Salmonella typhi* porins (Gonzales *et al*, 1995). Resistant to immunosuppression of contact hypersensitivity by ultraviolet B light (Noonan and Hoffman, 1994). Low neutrophil response to thioglycolate broth and killed bacteria (contrast C57BL/10) (Marley *et al*, 1994). Pristane induces immune complex glomerulonephritis in association with autoantibodies typical of lupus erythematosus, though the strain is not normally considered to be susceptible to the disease (Satoh *et al*, 1995). The IgE response following topical application has been used to predict which chemicals may have the potential to cause sensitization of the respiratory tract (Hilton *et al*, 1996). Diminished expression of neutral glycosphingolipid GgOse(4)Cer in concanavalin A stimulated T lymphoblasts (Muthing, 1997). The potential influence of circadian changes and laboratory routine on some immune parameters has been described by Kolaczowska *et al* (2000).

Infection

Highly susceptible to infection by *Salmonella typhimurium* strain C5 (Plant and Glynn, 1974; Robson and Vas, 1972). Relatively resistant to a natural intestinal helminth infection (Eaton, 1972). High susceptibility to BALB/Tennant leukemia virus (Tennant, 1965). Transmission of murine leukemia virus (Scripps) through three successive generations 100% (Jenson *et al*, 1976). Highly susceptible to development of leukemia on infection with Friend virus (Dietz and Rick, 1972). Susceptible to *Mycobacterium marinum* and good plateau harvest of *M. leprae* 8 months after infection (Shepard and Habas, 1967). Susceptible to infection with *Mycobacterium paratuberculosis*, and develops a chronic infection (Chiodini and Buerge, 1993). Susceptible to infection

with *Mycobacterium avium*, but resistance is enhanced by Freund's incomplete adjuvant (Castro *et al*, 1993). Susceptible to infection with *Yersinia enterocolitica* associate with a poor interferon gamma response (contrast C57BL/6) (Autenrieth *et al*, 1994). Susceptible to the induction of chronic pyelonephritis with *Escherichia coli* after introduction of the bacteria by the ascending route (Gupta *et al*, 1995).

Relatively resistant to infection with *Helicobacter felis* (contrast C57BL/6) (Mohammadi *et al*, 1996). Resistant to infection by *Helicobacter felis* with only mild gastritis in the antrum and no atrophy seen over time (cf CBA, contrast 4 other strains) (Sakagami *et al*, 1996). Susceptible to mouse hepatitis virus type 3 (Le Prevost *et al*, 1975). Resistant to mouse adenovirus type 1 (contrast C57BL/6) (Guida *et al*, 1995).

Resistant to induction of diabetes mellitus by encephalomyocarditis virus (Boucher *et al*, 1975; Hirasawa *et al*, 1995). Resistant to measles virus induced encephalitis, which correlates with a low cytotoxic T-lymphocyte response (contrast C3H, C57BL/6) (Niewiesk *et al*, 1993). Highly susceptible to the *Leishmania tropica* parasite, with the local disease being uncontrolled and with the development of metastases and fatal visceralization (Howard *et al*, 1980). Supported sustained growth of six strains of *Leishmania mexicana mexicana* (contrast C57BL/6) (Monroy-Ostria *et al*, 1994). Highly susceptible to *Leishmania major*, with the parasites disseminated within 10-24 hrs. from the site of subcutaneous footpad injection into the popliteal lymph node, spleen, lung, liver and bone marrow in contrast to resistant C57BL/6, CBA/J and C3H/HeJ (Laskay *et al*, 1995; Scott *et al*, 1996). Susceptible to infection with the helminth worm *Angiostrongylus*

costaricensis (Ishii and Sano, 1989). Susceptible to the induction of dental caries due to infection with *Streptococcus mutans* (Kurihara *et al*, 1991). Resistant to infection with *Pseudomonas aeruginosa* in contrast with susceptible DBA/2 mice (Morissette *et al*, 1995). Resistance is associated with a quicker inflammatory response and earlier initiation of bacterial clearance (Morissette *et al*, 1996). Develop mycotic mastitis following inoculation of the mammary gland with *Candida krusei* isolated from bovine mastitis (Guhad *et al*, 1995). Susceptible to the development of chronic Chagas' cardiomyopathy in postacute *Trypanosoma cruzi* infection (Rowland *et al*, 1992). Susceptible to infection with *Trypanosoma congolense* with unrestrained parasite growth to the time of death about 12 days later (contrast C57BL/6) (Ogunremi and Tabel, 1995). Resistant to lethal and body weight effects of *Toxocara canis*, but high larval brain levels (Epe *et al*, 1994). Infection with larval *Echinococcus multilocularis* by transportal injection of hyatid homogenate results in a multivesiculation form of hyatid development (Nakaya *et al*, 1997). Susceptible to *Streptococcus suis* type 2 including the type strain, two isolates from meningitis in pigs and two isolates from tonsils of clinically healthy pigs (Kataoka *et al*, 1991). Resistant to street rabies virus (SRV) injected via the intraperitoneal route (Perry and Lodmell, 1991). Following administration of murine cytomegalovirus, BALB/c, BALB.B, and BALB.K mice develop persistent myocarditis regardless of age

at infection, and age-related cardiopathy is frequent and severe in infected and uninfected mice (contrast C57BL/10 and C3H) (Price *et al*, 1991). Susceptible to the lethal effects of murine hepatitis virus strain 3 (contrast A/J) (Fingerote *et al*, 1995). The mouse hepatitis virus JHM strain induces a biphasic retinal disease (Wang *et al*, 1996). Susceptible to infection with the tick-born Thogoto virus, with severe symptoms and death after a few days. The congenic strain carrying the Mx1 gene from strain A2G is resistant (Haller *et al*, 1995). Susceptible to herpes simplex virus-1 (contrast C67BL/6) (Brenner *et al*, 1994). Develop carditis on infection with Lyme borreliosis (*Borrelia burgdorferi*) (Barthold *et al*, 1990), but develop only mild arthritis (contrast C3H/HeJ) (Matyniak and Reiner, 1995). Hepatic amoebiasis can be induced by introducing *Entamoeba histolytica* infected hamster liver tissue

in between the adjacent liver lobes of these mice. (Bhol *et al*, 1990). Resistant to intravaginally inoculated *Neisseria gonorrhoea* (Johnson *et al*, 1989). Susceptible to infection with *Ehrlichia risticii* (Williams and Timoney, 1994) Widely used in study of Plasmodium berghei infections, though much less sensitive than C57BL/6 (Scheller *et al*, 1994).

Infection with P. berghei results in high peripheral blood and death within 22-24 days, but without neurological complication, in contrast with the more susceptible C57BL/6 (Moumaris *et al*, 1995). Susceptible to disseminated *Cryptococcus neoformans* (Irokanulo and Akueshi, 1995). *Nippostrongylus brasiliensis* normally rejected

by 14 days post infection. However, this pattern of self-cure was not observed in a "putative" BALB/c substrain from the University of Texas (Mayberry *et al*, 1993). Susceptible, with high amylase response to the fungus *Paracoccidioides brasiliensis* (Xidieh *et al*, 1994). Susceptible to the protozoan parasite *Neospora canium* following subcutaneous inoculation with tachyzoites of the NC-1 strain (Lindsay *et al*, 1995). May develop Mite- associated ulcerative dermatitis with an allergic reaction to parasite- derived substances following infection with *Mycoptes musculus* (Jungmann *et al*, 1996). The composition of the oral bacterial population is influenced by the origin (supplier) of the animals (Rodrigue and Lavoie, 1995).

Life-span and Spontaneous Disease

The BALB/c mouse has a low mammary tumor incidence. Primary lung tumors in 2.5% of the animals. Transplantable medullary thyroid carcinoma (Van Zwieten *et al*, 1983). No correlation between the frequencies of benign monoclonal gammopathy and H-2 haplotype was found (Van den Akker *et al*, 1987). Median life-span 18.0 months in BALB/ cJ males and 19.7 months in BALB/cJ females (Storer, 1966). Median life-span 21.4 months in BALB/cJ males and 23.9 months in BALB/cJ females (Les, 1969). Median life- span 9.9 months in BALB/ cJ males and 14.9 months in BALB/cJ females (Les, 1966). Median life-span 13.2 months in BALB/cJ males and 20.2 months in BALB/cJ females (Ebbesen,

1971). Median life-span 13.2 months in BALB/c males and 20.2 months in BALB/c females (Ebbesen, 1971). Median life-span 17.0 months in BALB/cj males and 18.7 months in BALB/cj females (Festing and Blackmore, 1971). Median life-span 15.6 months in BALB/cj males and 20.3 months in BALB/cj females (Grahn, 1972). Median life-span 21.6 months in BALB/cj males and 27.2 months in BALB/cj females (Goodrick, 1975). Amyloidosis 40% in males. Reticular neoplasms 23% females and 3% males (Ebbesen, 1971). Primary lung tumors 32% in males, 30% in breeding females and 14% in virgin females in Scott substrain. Leukemia 5% (Myers *et al*, 1970). Zero incidence of lymphatic leukemia. Mammary adenocarcinomas zero in males, 5% in breeding females and 1% in virgin females (Hoag, 1963). Mammary tumors 30% at 2 years (Bentvelzen *et al*, 1970). Mammary tumors 20% in females at 16.7 months, but 100% at 7.1 months in BALB/cfC3H (Heston and Vlahakis, 1971). Mammary tumors 10% at 14 months (Schlom *et al*, 1973). Low gross tumor incidence in males (Storer, 1966). Renal tumors 25-48%, mammary tumors 3-13%, reticuloendothelial tumors 11-20%, lung tumors 10-16%, synoviomias 2-8%, depending on substrain (Sass *et al*, 1976). Low incidence of virus-like particles in chemically-induced sarcomas (Liebelt *et al*, 1970). Frequency of rhabdomyosarcomas was calculated to be 2.4/100,000 mice retained as breeders, and 10/14 mice found with these tumors were of the BALB/cj substrain (Sundberg *et al*, 1991a). No brain tumors in contrast with C3H (Morgan *et al*, 1984). Rare spontaneous myoepitheliomas arising from myoepithelial cells of various exocrine glands have been observed in the J and ByJ substrains (Sundberg *et al*, 1991b). Gross

tumor incidence in germ-free mice 43%, with lung tumors 21%, angiomas 6%, lymphosarcomas 5% and other tumor types less than 3% each (Smith and Pilgrim, 1971). Pulmonary tumors 26-29% (Heston, 1968).

Left auricular thrombosis occurs in 66% of older breeding females. This is associated with reduced levels of the prothrombin complex factors such as factor IX (40% of normal), factor XIII (60% of normal), factor X (50% of normal) and prothrombin (about 33% of normal).

These deficiencies occur slightly before parturition (Meier and Hoag, 1966). High incidence of epicardial mineralization (11%

in males, 4% in females), which increases slightly with age (Frith *et al*, 1975). Heart defects, including cardiac calcinosis 17-62% (Festing and Blackmore, 1971). Spontaneous myocardial lesions of right ventricle found in 60% of females and 30% of males. These macroscopically visible degenerative fibrosclerotic lesions may represent a last phase of myocarditis of the inflammatory type found in apparently normal mice (Bellini *et al*, 1976). BALB/c mice carry a single recessive gene different from that found in C57BL/6J and WB/ReJ, causing age-related hearing loss (Willott *et al*, 1995). The tumor incidence has been described by Dragani (1979). Uterine lesions have been described by Malinin and Malinin (1972). The relationship of genotype, sex, body weight, and growth parameters to lifespan in inbred and hybrid mice has been described by Ingram *et al* (1982). A review of the life span of aging mice has been described by Myers, (1978).

Miscellaneous

Recommended host for transplantable tumors: melanoma HP and pleomorphic sarcoma 5180, although the latter is not host-specific (Kaliss, 1972). Low mortality after neonatal thymectomy (Law, 1966). Embryonic stem cell lines have been established (Kawase *et al*, 1994). Characteristics of the BALB/c strain have been described by Festing (1997) and Lyon *et al*, (1996). The history and characteristics have been reviewed by Potter (1985).

Physiology and

Biochemistry High Na/K

ratio in erythrocytes (Waymouth, 1973). Low plasma cholinesterase activity in females (Angel *et al*, 1967). Low levels of serum ceruloplasmin in males (Meier and Macpike, 1968). Low serum haptoglobin level (Peacock *et al*, 1967). High plasma cholesterol levels (Jiao *et al*, 1990). High systolic blood pressure (Schlager and Weibust, 1967). Low mean heart rate but high heart rate adaptation (Blizard and Welty, 1971). High erythrocyte catalase level (Hoffman and Rechcigl, 1971). Low intra-ocular pressure (John, *et al*, 1997). High peripheral nerve conduction velocity (Hegmann, 1972).

High brain L-glutamic acid decarboxylase (GAD) and choline acetyltransferase and catechol-

Omethyltransferase; low brain acetylcholinesterase and monoamine oxidase activity (Tunncliff *et al*, 1973). High brain tyrosine hydroxylase activity (Ciranello *et al*, 1972). High brain plasmalogen (Sampugna *et al*, 1975).

High proportion of time spent sleeping with a high percentage of slow-wave sleep and low proportion of paradoxical sleep (Valatx and Bugat, 1974). Short tau DD, the endogenous (free-running) period of the circadian pacemaker measured in constant environmental darkness (Schwartz and Zimmerman 1990). High hypoxanthene- guanine phosphoribosyl transferase in the thalamus (Suran 1973). Low N-methylnicotinamide oxidase activity (Huff and Chaykin, 1967). Low rectal and tail temperature (Shepard and Habas, 1967). High kidney arylsulphatase activity (Daniel, 1976). Low basal level of serum prolactin (Sinha *et al*, 1975). Low

spermatzoal betagluturonidase activity (Erickson, 1976). Urine has high osmolarity (Silverstein, 1961). High basal levels of kidney catalase, and superoxide dismutase but low basal level of kidney glutathione peroxidase and kidney glutathione (Misra *et al*, 1991). High level of alpha-fetoprotein in amniotic fluid and neonatal plasma (Adinolf *et al*, 1990). High levels of alpha-fetoprotein in adult mice (Olsson *et al*, 1977). Low hepatic microsomal coumarin hydroxylase activity in males (Van Iersel *et al*, 1994). Secretory group II phospholipase A2 gene has very high expression in small intestine (contrast 129/Sv and C57BL/6) (Kennedy *et al*, 1995). Stress in mice after tail bleeding has been described by Tuli *et al* (1995a). Stress after transportation has been described by Tuli *et al* (1995b). Erythrocyte oxidative stress hemolysis is influenced by the presence of the *Hbbd* allele (Kruckeberg, 1991; Kruckeberg *et al*, 1987). High levels of alpha-fetoprotein in adult mice (Olsson *et al*, 1977).

Reproduction

The BALB/c has a good breeding performance and a long reproductive life- span. Colony output 1.18 young/female/ wk, litter size at weaning 5.2 (Festing, 1976). Good breeding performance, mean 3.24 young/ female/month (Hansen *et al*, 1973). Intermediate breeding performance, litter size 5.1, sterility 32% (Nagasawa *et al*, 1973). Low litter size (Verley *et al*, 1967). Low pre-implantation loss of embryos, but high post- implantation losses (Leonard *et al*, 1971). Embryos subject to the 2- cell block and only grow successfully in culture from the late 2-cell stage (Sekirina and Neganova, 1995).

between these two substrains. The Qa-2 gene is one gene that does differ between those substrains and involves a deletion in the BALB/cBy genome.

Three major substrains trace back to before 1940. Data on genetic markers suggest that

these substrains have diverged largely through mutation or residual heterozygosity rather than genetic contamination. (Hilgers *et al*, 1985) have shown that the substrains differ as a result of mutations at the Raf1 locus (controlling the expression of alpha- fetoprotein), the Qa2 locus (governing cell surface antigens), the Gdc1 locus (governing L-glycerol 3-phosphate dehydrogenase activity in the liver) and the PR1 repetitive sequence. There is no evidence for genetic contamination during the early history of the strain.

This strain carries the Mus musculus musculus Y-chromosome, while others have the M. m. domesticus type (Nishioka, 1987).

Immunology

Resistant to experimental allergic encephalomyelitis (EAE) (Levine and Sowinski, 1973). Resistant to EAE with short duration but moderate mortality (Lindsey, 1996). Description of an allergic model in BALB/c mice (Hessel *et al*, 1995a; Hessel *et al*, 1995b) where IL-16 is involved (Hessel *et al*, 1998). High lymphocyte phytohemagglutinin response (Heiniger *et al*, 1975). Good immune response to type III pneumococcal polysaccharide (Braley and Freeman, 1971). Good splenic PFC immune response to pneumococcal polysaccharide (Amsbaugh *et al*, 1972). Immune response of SJL mice to type-III pneumococcal polysaccharide declines by 42 weeks, in contrast to BALB/c and C3H (Smith, 1976). Poor primary immune response to bacteriophage fd (Kölsch *et al*, 1971). Poor immune response to synthetic doublestranded RNA (Steinberg *et al*, 1971). Responder to synthetic polypeptide (Pinchuck and Maurer, 1965) and Glu60, Ala30, Tyr10 (Dorf *et al*, 1974). Very good immune response to cholera A and B antigens (Cerny *et al*, 1971). Good immune response to

dextran -1,3-glucosyl
linkages (Blomberg

et al, 1972). High responder
to dextran (Blomberg *et al*,
1972). Good primary
immune hemolysin and
hemagglutinin response
(Ghaffar and James, 1973).
Poor immune response to
Salmonella anatum, S.
senftenberg and S.
strasbourg
lipopolysaccharide (Di Pauli,
1972). Good immune
response to Vi antigen
(Gaines *et al*, 1965).
Precipitating and skin-
antibodies have fast
electrophoretic mobility
(Fahey, 1965). Non-
discriminator between
“H” and “L” sheep RBC
(McCarthy and Dutton,
1975). Low anti-DNP
antibody concentration
(Paul *et al*, 1970). High
PHA- stimulated
lymphocyte blastogenic
response (Hellman and
Fowler, 1972).
Erythrocytes have a low
agglutinability (Rubinstein
et al, 1974). Resistant to
induction of experimental
autoimmune thyroiditis
(Vladutiu and Rose, 1971).
Resistant to induction of
autoimmune prostatitis
(contrast C57BL/6) (Keetch
et al, 1994). Immunization by
intraperitoneal injection of
fetal human (but not calf)
proteoglycan depleted of
chondroitin sulfate
together with complete or
incomplete Freund's
adjuvant produces
progressive polyarthritis
and ankylosing spondylitis.
Clinical assessment
suggests that affected mice
have many similarities to
human rheumatoid arthritis
and ankylosing spondylitis.
Eventually, the joints
become stiff and deformed.
Antibodies against
collagen type II were
detected in approximately
25% of
arthritic mice, but only
following cartilage
degradation. Sublines

differed in their response,

but 9 other mouse strains
and 5 F1 hybrids were
resistant. See Glant *et al*,
(1993) for a review. Resistant
to induction of anaphylactic
shock by ovalbumin
(Tanioka and Esaki, 1971).
Anti- BPO IgE monoclonal
antibody failed to produce
potent systemic
sensitization sufficient
for provocation of lethal
shock in most aged (6 to 10
months) mice (Harada *et al*,
The BALB/cJ and
BALB/cByJ were separated
in 1935 at F38. There are
very few genetic differences

BALB/c

BALB/cOlaHsd

6-8 weeks old, Females

non-fasted

Clinical Chemistry	n = 25	Units	Mean	Std Dev
Glucose		mmol/L	191.6	13.1
Blood Urea Nitrogen (BUN)		mg/dL	16.8	2.6
Creatinine		mg/dL	0.1	0.0
BUN/Creatinine		Ratio	132.3	
Cholesterol		mg/dL	112.7	10.0
Total Protein (TP)		g/dL	5.2	0.2
Albumin (Alb)		g/dL	2.3	0.1
Globulin (Glb)		g/dL	2.9	0.1
Alb/Glb		Ratio	0.8	
Total Bilirubin		mg/dL	0.2	0.1
Alkaline Phosphatase (ALP)		U/L	262.1	34.9
Aspartate Aminotransferase (AST)		U/L	191.5	141.2
Alanine Aminotransferase (ALT)		U/L	97.8	83.5
AST/ALT		Ratio	2.0	
Calcium (Ca)		mg/dL	9.0	0.2
Phosphorus (P)		mg/dL	8.9	0.6
Sodium (Na)		mmol/L	152.3	2.2
Potassium (K)		mmol/L	8.9	0.9
Chloride (Cl)		mmol/L	113.4	3.8

Hematology	n = 24	Units	Mean	Std Dev
White Blood Cells (WBC)		K/uL	11.0	1.7
Red Blood Cells (RBC)		M/uL	10.3	0.5
Hemoglobin (Hgb)		g/dL	16.7	0.9
Hematocrit (Hct)		%	52.9	3.0
Mean Corpuscular Volume (MCV)		fL	51.5	0.8
Mean Corpuscular Hemoglobin (MCH)		pg	16.3	0.2
Mean Corpuscular Hemoglobin Concentration (MCHC)		g/dL	31.6	0.3
Basophils		%	0.1	0.1
Neutrophils		%	11.4	1.3
Lymphocytes		%	84.1	1.7
Monocytes		%	3.3	1.1
Eosinophils		%	1.2	0.4

BALB/c

BALB/cOlaHsd

6-8 weeks old, Males

non-fasted

Clinical Chemistry	n = 25	Units	Mean	Std Dev
Glucose		mmol/L	211.0	18.5
Blood Urea Nitrogen (BUN)		mg/dL	19.7	3.2
Creatinine		mg/dL	0.1	0.0
BUN/Creatinine		Ratio	194.9	
Cholesterol		mg/dL	127.1	6.0
Total Protein (TP)		g/dL	5.2	0.2
Albumin (Alb)		g/dL	2.3	0.1
Globulin (Glb)		g/dL	2.9	0.1
Alb/Glb		Ratio	0.8	
Total Bilirubin		mg/dL	0.1	0.0
Alkaline Phosphatase (ALP)		U/L	257.3	47.6
Aspartate Aminotransferase (AST)		U/L	108.7	44.0
Alanine Aminotransferase (ALT)		U/L	78.9	34.1
AST/ALT		Ratio	1.4	
Calcium (Ca)		mg/dL	9.2	0.2
Phosphorus (P)		mg/dL	9.9	0.7
Sodium (Na)		mmol/L	154.9	1.9
Potassium (K)		mmol/L	9.6	0.5
Chloride (Cl)		mmol/L	117.3	2.6

Hematology	n = 25	Units	Mean	Std Dev
White Blood Cells (WBC)		K/uL	10.3	1.7
Red Blood Cells (RBC)		M/uL	10.3	0.4
Hemoglobin (Hgb)		g/dL	16.5	0.6
Hematocrit (Hct)		%	53.7	2.0
Mean Corpuscular Volume (MCV)		fL	52.0	0.7
Mean Corpuscular Hemoglobin (MCH)		pg	15.9	0.2
Mean Corpuscular Hemoglobin Concentration (MCHC)		g/dL	30.7	0.4
Basophils		%	0.0	0.1
Neutrophils		%	12.0	7.1
Lymphocytes		%	84.1	6.8
Monocytes		%	2.8	1.2
Eosinophils		%	1.0	0.4

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