



CBA

Origin

Developed in 1920 by Strong from a cross of Bagg albino female and DBA male. Strain C3H also originated from this cross. Strain CBA was selected for a low mammary tumor incidence and C3H for a high incidence.

CBA/CaOlaHsd

In 1932, to Haldane and Grüneberg, Department of Genetics, University College, London, UK. Then to Carter via Royal Cancer Hospital, London and British Empire Cancer Campaign, London in 1947. To Atomic Energy Research Establishment, Harwell circa 1953. Obtained by Laboratory Animal Centre, Carshalton from Atomic Energy Establishment, Harwell in 1956 and then to OLAC (now Envigo).

CBA/JCrHsd

From Jackson Laboratories, Bar Harbor, Maine, to National Cancer Institute, Frederick, Maryland in 1983. To Harlan Laboratories in 1987. Harlan became Envigo in 2015.

Research applications

Hearing, behavior, tumors, immunology, infections, reproduction.

Characteristics

The CBA mouse is widely distributed, and used as a general-purpose strain. There are several substrains recognized, and the differences between these substrains are probably too large to be accounted for by mutation.

Anatomy

Low brain weight (Storer, 1967). Large forebrain volume and neocortex (Wimer *et al*, 1969). Small brain/body weight ratio (Roderick *et al*, 1973). Low testes weight (Shire and Bartke, 1972). Large

kidney/body weight ratio (Schlager, 1968). Low thyroid weight (Mendoza *et al*, 1967). Small heart/body weight ratio (Mokler and Iturrarian, 1973). Large pituitary (Sinha *et al*, 1975). Low total leukocyte count, high erythrocyte count (Russell *et al*, 1951). Few bristles on foot pads (Festing, 1974). Third molars small and one or more absent in about 18% of the individuals (Hummel *et al*, 1966). Inner ear morphology in CBA/Ca mice in relationship to noise and age has been described by Hultcrantz and Li (1993).

Behavior

Low spontaneous bar-pressing activity, low tail rattling during aggressive encounters, high social grooming during aggressive encounters, low intrasrain aggression (Southwick and Clark, 1966). Low locomotor activity (Davis and King, 1967). Low locomotor activity when single, but intermediate when grouped (Davis *et al*, 1967). Low spontaneous locomotor activity (Nikulina *et al*, 1991). Low shock avoidance learning (Bovet *et al*, 1966; Bovet *et al*, 1969). High shock avoidance learning in Ca substrain but not in J substrain (Wahlsten, 1973). Highly susceptible to "pinch-induced" catalepsy (excessive freezing), possibly due to a single recessive autosomal gene (Kulikoff *et al*, 1993).

Drugs

Resistant to urethane-induced lung tumors (Falconer and Bloom, 1962). Susceptible to skin ulceration by DMBA (Thomas *et al*, 1973). Susceptible to induction of leukemia and liver tumors by neonatally administered DMBA (Flaks, 1968).

Susceptible to X-irradiation (Roderick, 1963), but resistant to 'CNS syndrome' with high doses of X-irradiation (Yugas, 1968). Susceptible to hyperbaric oxygen, showing central nervous system manifestations (Hill *et al*, 1968). Sensitive to lethal effect of ozone (Goldstein *et al*, 1973), but resistant to ozone-induced decreases of tracheal potential (Takahashi *et al*, 1995). Sensitive to teratogenic effect

of acetazolamide (Green *et al*, 1973; Hackman and Hurley 1983), but resistant to induction of cleft palate in embryos by cortisone (Kalter, 1965). Insensitive to insulin (Brown, 1965). Long survival on Warfarin (Lush and Arnold, 1975). High ED50 to behavioral effects of nicotine (Marks *et al*, 1989). Susceptible to weight loss induced by cocaine, but this is attenuated by anisomycin (cf C3H, SJL) (Shimosato *et al*, 1994). More resistant to acute toxic effects of aflatoxin B-1 than strain C57BL/6 (Almeida *et al*, 1996).

Genetics

Coat color genes - A, B, C, D : agouti (wild type).

Histocompatibility - H-2k.

Biochemical markers:

CBA/CaOlaHsd - *Apoa-1^a*, *Car-2^a*, *Es-1^b*, *Es-2^b*, *Es-3^c*, *Gpd-1^b*, *Gpi-1^b*, *Hbb^d*, *Idh-1^b*, *Ldr-1^b*, *Mod-1^b*, *Pep-3^b*, *Pgm-1^b*, *Trf^e*.

CBA/JCrHsd - *Apoa-1^b*, *Car-2^b*, *Es-1^b*, *Es-2^b*, *Es-3^c*, *Gpd-1^b*, *Gpi-1^b*, *Hbb^d*, *Idh-1^b*, *Ldr-1^a*, *Mod-1^b*, *Pep-3^b*, *Pgm-1^a*, *Trf^e*.

Skin grafts between CBA/Ca and CBA/J are rejected. (Green and Kaufer, 1965). Genetic polymorphism within CBA sublines has been described by Whitmore and Whitmore (1985). This strain carries the *Mus musculus musculus* Y-chromosome, while others have the *M. m. domesticus* type (Nishioka, 1987).

Immunology

Low lymphocyte phytohemagglutinin response (Heiniger *et al*, 1975). Good immune response to low doses of bovine gamma-globulin (Levine and Vaz, 1970). Poor splenic PFC immune response to pneumococcal polysaccharide in CBA/J and CBA/H (Amsbaugh *et al*, 1972). Good immune response to ovomucoid, but poor response to ovalbumin (Vaz *et al*, 1971). Resistant to induction of antigen-induced arthritis (contrast most other strains). (Brackertz *et al*, 1977). Non-responder to synthetic polypeptide Glu⁵⁷, Lys³⁸, Ala⁵ (Pinchuck and Maurer, 1965). Discriminator between 'H' and 'L' sheep red blood cells (McCarthy and Dutton, 1975). Erythrocytes have a high agglutinability (Rubinstein *et al*, 1974). Low responder to dextran (Blomberg *et al*, 1972). Low immune response to ganglio-series gangliosides (Kawashima *et al*, 1992). High natural killer cell response to the immuno-stimulant 7-allyl-8-oxoguanosine (Pope *et al*, 1994).

Diminished expression of neutral glycosphingolipid GgOse(4)Cer in concanavalin A stimulated T lymphoblasts in J substrain (Muthing, 1997). Long peptides have the advantage of covering all potential T cell epitopes, and may represent novel and safe tools for the therapy of allergic diseases (Astori *et al*, 2000).

Infection

Resistant to infection by *Salmonella typhimurium* strain C5 (Plant and Glynn, 1974). Susceptible to infection by liver fluke *Opisthorchis felineus* (Zelentsov, 1974). Good plateau harvest of *Mycobacterium leprae* 8 months after infection (Shepard and Habas, 1967). Low immune response to *Mycobacterium lepraemurium* is associated with an impaired macrophage function (Saito and Natori 1985). Resistant to infection by *Helicobacter felis* with only mild gastritis in the antrum and no atrophy seen over time (cf BALB/c, contrast 4 other strains) (Sakagami *et al*, 1996). Resistant to induction of diabetes mellitus by encephalomyocarditis virus (Boucher *et al*, 1975). Highly susceptible to measles virus (Rager-Zisman *et al*, 1976). Ca and N substrains carry no detectable endogenous ecotropic MuLV DNA sequences (Jenkins *et al*, 1982).

Administration of cadmium results in a high incidence of activation of latent herpes simplex virus infections. This is not seen in other strains, and does not correlate with cadmium toxicity (Fawl *et al*, 1996). J substrain resistant to the induction of dental caries due to infection with *Streptococcus mutans* (Kurihara *et al* 1991). Resistant to murine herpes virus (Kapoor *et al* 1992). Resistant to intra-vaginally inoculated *Neisseria gonorrhoeae* (Johnson *et al*, 1989). Develop severe lesions following infection with *Candida albicans* (Ashman *et al*, 1993). This correlates with low induction of *Candida*-specific gamma interferon (Ashman and Bolitho, 1993). CBA/J resistant, with low amylase response to the fungus *Paracoccidioides brasiliensis* (Xidieh *et al*, 1994). J substrain is susceptible to several clinical isolates of penicillin-susceptible and resistant strains of *Streptococcus pneumoniae*, and may be a useful model for evaluating antibiotic efficiencies against this bacterium (Tateda *et al*, 1996).

Resistant to *Leishmania major* (contrast BALB/c) (Laskay *et al*, 1995). Ca substrain susceptible to *L. major mexicana*, and vaccination against the parasite using liposomes with parasite membrane antigens was effective (cf C57BL/6 but contrast C57BL/10) (Lezama-Dávila, 1997). Infection with larval *Echinococcus multilocularis* by transportal injection of hyatid homogenate results in well-developed protoscoleces (Nakaya *et al*, 1997).

Life-span and spontaneous disease

Median life-span in CBA/J mice for both sexes 17.6 months (Storer, 1966). Median life-span 16.2 months in CBA/Ca males and 27.5 months in CBA/Ca females. Short median life-span of males associated with a high incidence of hemothorax, suggesting a high sensitivity to vitamin K deficiency in SPF conditions (Festing and Blackmore, 1971). Median life-span 25.0 months in CBA/J males and 21.2 months in CBA/J females (Smith *et al*, 1973). Median life-span 30.4 months in CBA/ males (Sharp *et al*, 1975).

High gross tumor incidence (Storer, 1966). Overall tumor incidence 29% in males, 55% in females,

including lymphoma 6% in males, 15% in females, hepatoma 24% in males, zero in females and mammary tumors 33% in females and zero in males (Smith *et al*, 1973). Lung adenomas 2-11% in BrA substrain, leukemia 4-10% (Muhlbock and Tengbergen, 1971). Resistant to the induction of atherosclerosis by a high-fat and high-cholesterol diet (Roberts and Thompson, 1976). Develop a mild hearing loss with onset late in life (contrast C57BL/6J) (Li, 1992, Willott *et al*, 1993, Li *et al*, 1993, Li and Borg, 1993). Do not carry any of the single recessive genes found in BALB/cBy, C57BL/6 and WB/ReJ, causing age-related hearing loss. All three genes are present in DBA/2 (Willott *et al*, 1995). The musculature of aging lungs has been studied by Heppleston (1961). The occurrence of epithelial and non-epithelial tumors in aging mice is described by Kawada and Ojima (1977).

The J substrain is carrier of the retinal degeneration (*Pde6b^{rd1}*) mutation.

Miscellaneous

Recommended host for transplantable rhabdomyosarcoma BW 10139 (Kaliss, 1972). High rate of spontaneous mutations (Schlager and Dickie, 1967). Characteristics of the CBA strain have been described by Festing (1997) and Lyon *et al*, (1996).

Physiology and biochemistry

High systolic blood pressure (Schlager and Weibust, 1967). Low Na/K ratio in erythrocytes and in plasma (Waymouth, 1973). High serum ceruloplasmin levels in males of Ca and J substrains, high level in females of Ca substrain but intermediate in J substrain (Meier and MacPike, 1968). Low calcium uptake by the heart (Mokler and Iturrian, 1973). High proportion of the time spent sleeping, with large percentage of slow-wave sleep and low percentage of paradoxical sleep (Valatx and Bugat, 1974). Low percentage of paradoxical sleep (Pagel *et al*, 1973). Low metabolic rate according to Storer (1967), but high metabolic rate according to Pennycuik (1967). High cell turnover in J substrain as estimated by rapid clearance of

DNA-bound radioactivity (Heiniger *et al*, 1972). High peripheral nerve conduction velocity (Hegmann, 1972). High brain glutamic acid decarboxylase (Gaitonde and Festing, 1976).

Low hypoxanthine-guanine phosphoribosyl transferase in thalamus, but high level in hypothalamus (Suran, 1973). High brain monoamine oxidase and low level of catechol-O-methyltransferase activity (Tunncliffe *et al*, 1973). Low brain tyrosine hydroxylase activity (Ciranello *et al*, 1972). Low peptidyl proline hydroxylase activity in mammary gland, foot pad and tumors (Cutroneo *et al*, 1973). High sensitivity to thyrotropin in J substrain (Levy *et al*, 1965). High coumarin hydroxylating ability (Lush and Arnold, 1975). High hepatic 3 aminolevulinic acid synthetase activity after DDC treatment (Gross and Hutton, 1971). High porphyrin content in Harderian gland (Margolis, 1971). High hind foot pad temperature (Shepard and Habas, 1967). Have only about 20% of the maltase (gamma-glucoamylase) activity found in other strains, though there is no evidence for any gross metabolic abnormality resulting from this defect (Quezada-Calvillo *et al*, 1993). The adrenal and renal functions have been described by Shire (1968).

Reproduction

Good breeding performance, colony output 1.15 young/female/week, litter size at weaning 5.8, (Festing 1976). Intermediate breeding performance, litter size 5.4, sterility 5.2% (Nagasawa *et al*, 1973). Good litter size, depending on parity), but low proportion of females produce four or more litters (Fernandes *et al*, 1973). Low percentage pre-implantation loss of embryos (Leonard *et al*, 1971). Females have a high rate of fetal resorption when mated with DBA/2 males, but this can be dramatically reduced by immunization with BALB/c, but not DBA/2J spleen cells. This may provide an animal model for the prevention of fetal death by vaccination (Chaouat *et al*, 1985). Maturity of the cytoplasm in oocytes is acquired earlier than in those of the KE or other strains of mice so far studied. (Polanski 1990)

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