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ENVIGO

Research Models
and Services

Inbred Rats

LEW (Lewis)

Origin

Inbreeding of the Lewis rat is begun by Dr. Margaret Lewis from a Wistar stock. In 1924, at F20 to Aptekman and Bogdon. In 1958, at F31 to Silvers, who distributed this strain subsequently.

LEW/Han[®]Hsd

To Central Institute for Laboratory Animal Breeding, Hannover, in 1973 at F58. In 1994, to Harlan Netherlands through acquisition of Central Institute for Laboratory Animal Breeding. Harlan became Envigo in 2015

LEW/SsNHsd

From National Institute of Health, Bethesda, Maryland, to Harlan Sprague Dawley, Inc. (now Envigo).

Characteristics

The Lewis rat is used as the inbred partner for a number of congenic strains at the major histocompatibility complex (Stark and Kren, 1969). The Lewis rat is sensitive to the development of a number of autoimmune diseases, including adjuvant-induced arthritis (Perlík and Zideck, 1974), experimental allergic encephalomyelitis (EAE) (Perlík and Zideck, 1974; Gasser *et al*, 1975; Willenborg, 1979), induced autoimmune myocarditis (Friedman *et al*, 1970), allergic adrenalitis (Andrada *et al*, 1968), allergic orchitis (Levine and Sowinsky, 1970), experimental autoallergic sialadenitis (a model of Sjogren's disease) (Cutler *et al*, 1987), and experimental autoimmune myasthenia gravis (Lennon *et al*, 1975). The LEW/HanHsd is very susceptible to the induction of EAE, while the LEW/SsNHsd is not susceptible to the induction of EAE. Neonatal and weanling rats susceptible to *Borrelia burgdorferi*-induced arthritic lesions resembling those found in human Lyme disease (Barthold *et al*, 1988). High hepatic metabolism of ethylmorphine in females (Page and Vesell, 1969). Low blood pressure, reaching 119 mmHg at ten weeks of age (Tanase *et al*, 1982).

Liver gangliosides are of the a-type (Kasai *et al*, 1993). Glomerular filtration rate and renal plasma flow described by Hackbarth (1981). Haematological parameters and their relation to diet have been described by Hackbarth (1983). Short gestation period: $22.43 \pm .22$ days (Peters, 1986).

Genetics

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| Coat colour genes | - <i>a, B, c, h</i> : albino. |
| Histocompatibility | - <i>RT1^l, RT2^a, RT3^a, RT7^a, RT8^b</i> . |
| Biochemical markers | - <i>Acon-1^b, Acp-2^a, Ahd-2^c, Akp-1^a, Alb^a, Amyl^a, Cryg-1^a, Es-1^a, Es-2^d, Es-3^d, Es-4^b, Es-6^a, Es-7^b, Es-8^a, Es-9^c, Es-10^b, Es-14^b, Es-15^b, Es-16^b, Es-18^a, Fh-1^a, Gc^a, Glo-1^a, Gox-1^a, Hbb^b, Igk-1^a, Lap-1^b, Mgd-1^b, Mup-1^b, Pep-3^a, Pg-1^a, Pgd^b, Svp-1^b</i> . |

Reproduction

High rate of sterility.

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