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

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 ± .22 days (Peters, 1986).

Genetics
Coat colour genes - a, B, c, h : albino.
Histocompatibility - RT1, RT2, RT3, RT7a, RT8b.
Biochemical markers - Acon-1p, Acp-2a, Ahd-2a, Akp-1a, Alb1, Amy1, Cryg-1a, Es-1a, Es-2a, Es-3a, Es-4a, Es-6a, Es-7a, Es-8a, Es-9a, Es-10a, Es-14a, Es-15a, Es-16a, Es-18a, Fl-1a, Gc1, Glo-1a, Gox-1a, Hbb1, Igk-1a, Lap-1a, Mgd-1b, Mup-1b, Pep-3a, Pg-1a, Pgd1a, Svp-1a.

Reproduction
High rate of sterility.
References


