

SS

Origin

John P. Rapp, Medical College, Ohio, from a colony developed by LK Dahl, Brookhaven National Laboratories, Upton, New York, who produced, a colony of rats sensitive to sodium chloride-induced hypertension by selection from outbred Sprague Dawley stock (Dahl et al, 1962).

SS/JrHsd

From Medical College to Harlan Laboratories in 1986. Harlan became Envigo in 2015.

Characteristics

Fulminant hypertension and marked vascular and renal lesions develop after three-four weeks on a high salt (8% NaCl) diet, and all die within eight weeks (Rapp and Dene, 1985), though on a low (0.3% NaCl) salt diet they survive well though they all develop marked hypertension, but with a slower rise in blood pressure. A high sucrose (52%) diet also leads to elevation of blood pressure, which is more rapid in SS than in SR or F344 rats (Preuss et al, 1992). Levels of IgG but not IgM or IgA are decreased in young pre-hypertensive rats compared with SR, and renal

dysfunction is manifested by increased excretion of high molecular weight proteins including albumin and immunoglobulins (Cowen et al, 1991). Blood pressure is also increased by dietary potassium (Rapp, 1984). Kidney transplantation experiments between SS and SR rats suggest that part of the sensitivity is due to the kidney, and SS rats have 15% fewer glomeruli than SR rats (Rapp, 1984). The inheritance of salt-dependent hypertension has been studied in crosses with SR by Abbot and Schachter (1994). SS rats develop a high frequency of cataracts, which may be associated with blood pressure response to sodium chloride (Estape et al, 1995).

Characteristics of the SS strain have been described by Greenhouse et al (1990).

Genetics

Coat color genes - c : albino.

Stlezin *et al* (1992) found that SS and SR had about 80% of DNA fingerprint bands in common.

Reproduction

Produces one litter of about 8 pups, but second litter smaller because of hypertension.

References

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