Overview

Also Known As: R3

This strain is no longer available as it has become extinct.

RIII S/J mice have prolonged bleeding times with normal platelet activity and low levels of factor VIII:C and plasma von Willebrand factor antigen, making it a good animal model for human von Willebrand disease. RIII S/J mice also produce a low antibody response to several bacterial polysaccharide antigens and are reported to be resistant to collagen induced arthritis. RIII S/J mice carry a spontaneous mutation, ldis1, that leads to a partial or complete disruption of the lens and cataract formation.
This strain is currently unavailable due to replenishing of cryopreserved stocks.
RIII/J mice have prolonged bleeding times with normal platelet activity and low levels of factor VIII:C and plasma von Willebrand factor antigen, making it a good animal model for human von Willebrand disease. This bleeding tendency is an incomplete dominant, autosomal trait. RIII/J mice also produce a low antibody response to several bacterial polysaccharide antigens and are reported to be resistant to collagen induced arthritis. Despite a B cell immunodeficiency, RIII/J mice develop severe experimental autoimmune myasthenia gravis (EAMG) (Tuzun et al., 2004).
RIII/J have a high incidence of mammary tumors and ovarian tumors. RIIIIS/J mice carry Mtv8, and Mtv14, but high incidence of tumors has not been reported in these mice. In fact, some studies indicate a resistance to chemically induced tumors. RIIIIS/J mice have been reported to develop far fewer lung tumors than A/J or SWR/J mice subsequent to Urethan treatment. BALB/c x RIII F1 males are also highly resistant to diethylstilbestrol-cholesterol induced testicular tumors even though BALB/c is highly susceptible. RIIIIS/J mice carry a spontaneous mutation, ldis1, that leads to a partial or complete disruption of the lens and cataract (Jablonski M, et al, 2004).
Appearance
albino
Related Genotype: \textit{Tyr}^{c}/\textit{Tyr}^{c}

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