Is There a Link between Resistance of Rats to Dextran and their Failure to Produce Reaginic Antibodies?

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Wistar rats selectively bred for non-reactivity to dextran have been found to produce few reaginic antibodies to allergens, such as ovalbumin and horse serum. Three criteria were used to distinguish IgE from other homocytotropic antibodies: (1) thermolability (instability at 56 °C for 4h); (2) sensitivity to sulphydryl agents (inactivation by mercapto-ethanol); and (3) persistence in passively sensitised skin (activity retained for up to 10 days). Titration of reaginic serum by PCA tests was carried out by intradermal injections of 0.1 ml of saline dilutions, the intravenous antigen and azovan blue dye being given 72 h later. The titre was expressed as the reciprocal of the greatest dilution giving a reaction greater than 5 min. Sensitisation was by ovalbumin (10 mg) or horse serum (1 ml), each mixed with Bordetella pertussis vaccine, followed 10 days later by a further dose of vaccine 4 days before estimating the reaginic titre.

Whereas most Wistar rats possessed titres of 64-128 and gave strong local cutaneous anaphylactic reactions when tested with intradermal allergen, Wistar rats selectively bred for non-reactivity to dextran (an autosomal recessive gene) had titres of 2-4 and gave very weak local reactions. On intravenous challenge with allergen, however, anaphylactic mortality in groups of 10 rats was similar (between 85 and 95%) although rats with low reaginic titres (those selectively bred for non-reactivity to dextran) died much later, often up to 24 h after challenge. Most Wistar rats respond to the first injection of clinical dextran (molecular weight 110,000) with the anaphylactoid reaction which resembles that occurring in allergic patients after the injection of or contact with the specific allergen.

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The signs and symptoms of the dextran reaction are mediated by released histamine and 5-hydroxytryptamine. Procedures such as treatment with insulin or with thyroxine potentiate the reaction and potentiate systemic anaphylaxis in these animals. Yet Wistar rats selectively bred for non-reactivity to dextran are unaffected by these procedures and are relatively resistant to heat shock (involving kinins) and arthritis induced by adjuvant. Recently, their isolated peritoneal mast cells have been found to release no histamine when incubated with dextran, even in the presence of phosphatidyl serine. Further, dextran reactivity has not so far been transferable to these resistant animals.

So, non-reactivity to dextran conveys upon the rat the ability to resist shock procedures and may be the reason for the failure to produce reaginic antibodies to certain allergens.