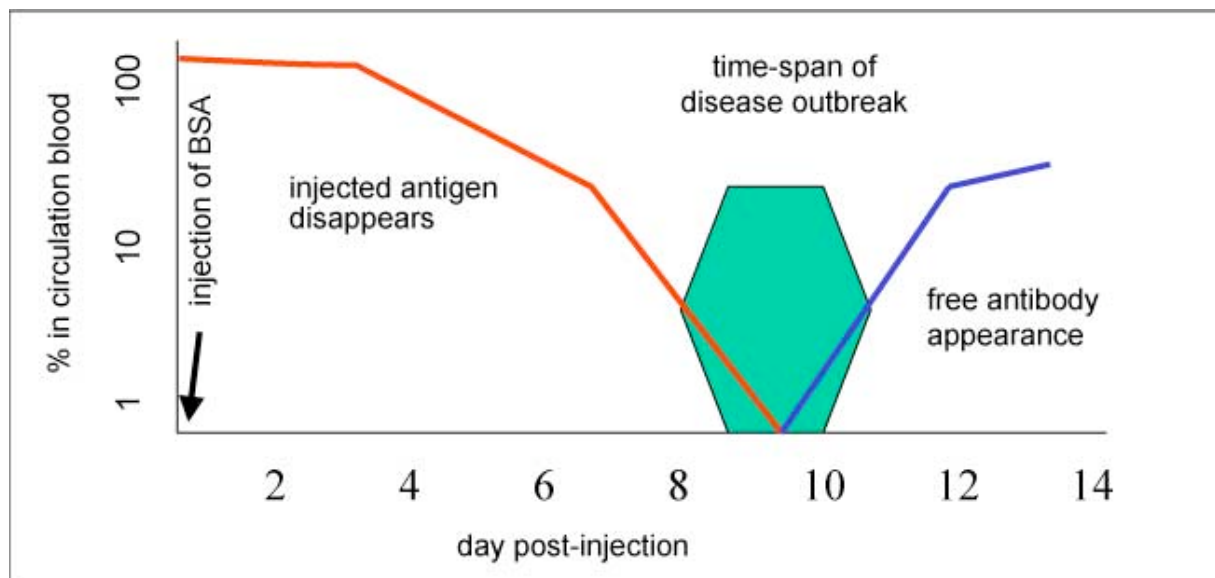


Serum Sickness

Serum sickness is a type III hypersensitivity reaction that develops after exposure to xenogeneic proteins. First described by von Pirquet and Schick in 1905: they see rash, fever, lymphadenopathy and arthralgia in recipients of horse serum for diphtheria. The pathogenesis of this serum sickness was attributed to the host's immunological reaction to injected antigens and has been described as a generalized Arthus reaction. Lambert and Dixon have conducted in the 1960ies several elegant animal studies using radiolabelled serum proteins conclusively demonstrating the association of the pathological lesions of serum sickness with the detection of circulating immune complexes (CIC). CIC and tissue bound complexes for at the time of immune elimination: for a number of factors determine whether CIC are deposited in tissue rather than cleared by the reticuloendothelial system. These factors include vascular permeability, local hyperdynamic flow, size of complexes and affinity of antibody. Distinction between acute and chronic serum sickness for understanding human immune complex disease is not useful because both forms present with a similar clinical picture - quite often an acute form precedes transformation into the chronic form with depletion of complement, morbilliform rash starting in the extremities and evolving with arthralgia, arthritis, systemic vasculitis and glomerulonephritis.



"The experimental model of serum sickness, usually studied with rabbits, has been developed by Lambert, Germuth and Dixon. It serves a good model for serum sickness in man: injection of 250 mg/kg radiolabelled antigen, e.g. bovine serum albumin (BSA) i.v., is followed by decrease in serum concentration (—). Soon, proteinuria develops (green surface) because of appearance of anti-BSA antibody that form circulating immune complexes, BSA/anti-BSA. In case of animal survival, free anti-BSA appears in the circulation (—)."