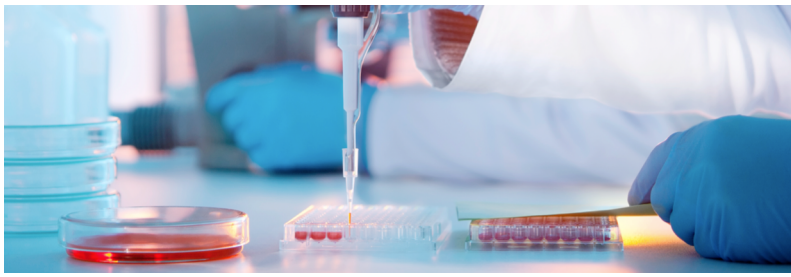


Anti-glomerular basement membrane (GBM) model in mice

Model Description

Anti-GBM glomerulonephritis is a well-established antibody-induced glomerulonephritis model in which mice are immunized with autologous or heterologous anti-GBM antibodies to mimic human nephritis via the initiation of an autoantibody-mediated inflammatory response. This model shares a number of pathogenic mechanisms with human glomerulonephritis, including immune complex deposition, complement activation and immune cell infiltration. To investigate the mechanisms of autoimmunity and inflammation in glomerulonephritis and to test approaches to specific immune intervention, we can induce anti-GMB disease in 2 mouse strains (C57Bl.6 and DBA.1 mice) using Sheep Anti-Rat Glomeruli (GBM) Serum (PTX-001) from Probetex or Rabbit anti-Mouse GBM from Lampire Bio labs to achieve a heterologous or autologous anti-GBM nephritis model.

Anti-GBM glomerulonephritis in mice

The GBM disease is characterized by immune-mediated glomerular inflammation. Most severe and rapidly progressive forms of glomerulonephritis feature the participation of autoantibodies mediating an inflammatory response in kidneys´ glomeruli. GBM is a highly reproducible model that progresses into a severe glomerular disease.

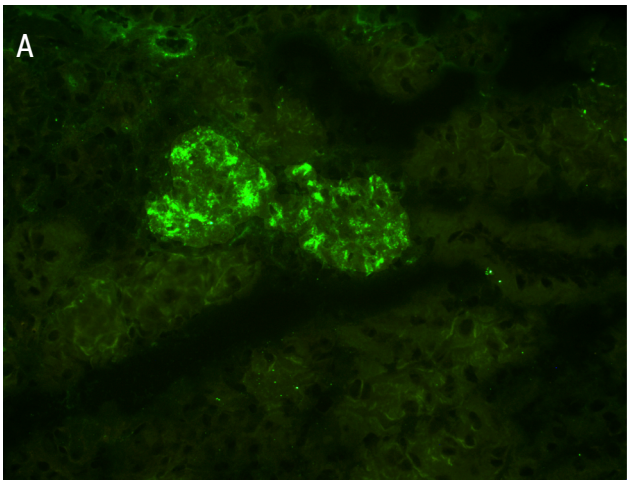
Strain	C57BL/6 or DBA.1
Induction	anti-GBM antibodies. Intravenous
Duration	20 days
Onset	2-3 days
Readouts	- Creatinine in blood and urine. - Histology (tissue damage, cellular infiltration)
Hystology	anti-C3, C3b/iC3b/C3c and C3d for kidney glomeruli sections.
Additional	Metabolic cages

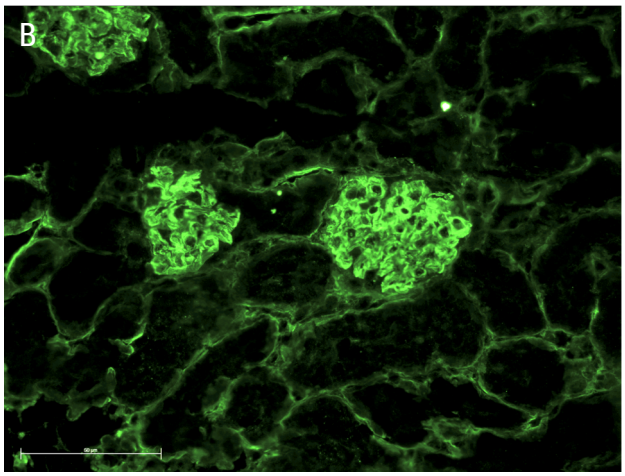
Characteristics

GBM disease is a rare autoimmune disorder characterized by the presence of circulating and deposited anti-GBM antibodies and a rapid progression of glomerulonephritis. The kidneys pathology is induced by an intravenous injection in the tail vein, with anti-GBM antibodies.

During the 18 days of the experiment, the animals undergo measurements in metabolic cages (e.g urine collection for assessments of urine metabolites) and weight assessment, as a general health parameter.

GBM disease induces glomerulonephritis with hyperplasia in glomeruli and increased mesangial cells and vasculitis with perivascular cell infiltration, glomerular deposition of IgG and complement C3. At study termination, the kidneys are collected for immunohistochemical analysis on markers like anti-C3, C3d and C3b/iC3b/C3c.





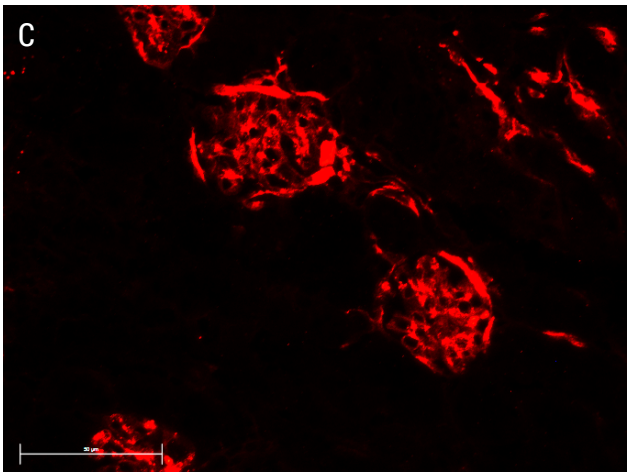


Figure 1. Fluorescence immunohistochemistry for autoantibodies deposition in kidneys` glomeruli: (A) C3 antibody; (B) C3d antibody and (C) C3b/iC3b/C3c antibody. Scale bar 50µm.

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Read more:

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