Advance SLE agent development with robust \textit{in vivo} models

Progress your systemic lupus erythematosus (SLE) agents to the clinic through our preclinical drug development platform. Utilize robust, spontaneous SLE models which more closely represent human disease.

- Select preclinical models with diverse mechanisms capturing many key characteristic clinical and pathologic features of SLE including:
  - MRL/Fas\textsuperscript{\textsubscript{lpr}} spontaneous model
  - NZB/W spontaneous model.
- Determine efficacy and response to treatment.
- Select qualified lupus lead agents.

**MRL/Fas\textsuperscript{\textsubscript{lpr}} spontaneous model of SLE**
- Spontaneous onset starting at 9-10 weeks of age, developing over 7-9 weeks.
- Major phenotype/characteristics:
  - dramatic lymphoproliferation and lymphadenopathy
  - splenomegaly
  - glomerulonephritis (subacute, proliferative) and proteinuria
  - skin lesions
  - expansion of CD3\textsuperscript{+}CD4\textsuperscript{-}CD8\textsuperscript{-} T cells
  - no interferon signature.

**NZB/W spontaneous model of SLE**
- Spontaneous onset starting around 22-23 weeks of age, developing over 12-13 weeks.
- Major phenotype/characteristics:
  - splenomegaly
  - glomerulonephritis (subacute, proliferative) and proteinuria
  - persistence of long-lived autoreactive plasma cells
  - interferon signature.

**Major endpoints for assessment:**
- Body weight.
- Skin lesions (in MRL/Fas\textsuperscript{\textsubscript{lpr}}).
- Proteinuria.
- Spleen, kidney, and lymph node size/weight.
- Cytokine levels.
- Blood serum anti-dsDNA antibody levels.
- Blood urea nitrogen (BUN) levels.
- FACS analysis of immune cell populations.
- Kidney histopathology and IHC staining for C3 and IgG levels.

\textbf{Urine Protein Levels and Kidney Weight of MRL/MpJ-Fas\textsuperscript{\textsubscript{lpr}} Mice}

\textbf{Kidney IgG Staining in the NZB/W Spontaneous Model of Lupus}

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