



# Lupus and Pregnancy

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## Technology description

PD-L1 Pathway in prevention of Lupus pregnancy complication

## Technology Overview

The uncontrolled inflammation of systemic lupus erythematosus (SLE) can cause severe pregnancy complications that threaten both mother and fetus, but most SLE treatments that suppress the immune system are considered unsafe during pregnancy. The increased rates of preeclampsia, pre-term birth and intra-uterine growth restriction in SLE pregnancy are only partially explained by the vascular effects of anti-phospholipid antibodies.

Led by Dr. Anne Stevens, the Stevens Lab is testing novel therapeutic agents targeting proteins that could regulate the immune system during pregnancy without suppressing it. The basis for these studies is that maternal-fetal tolerance during gestation relies on the same immunoregulatory molecules that are important in preventing autoimmunity.

One of these costimulatory molecules, PD-L1, inhibits allo- and autoreactive T cells through the receptor PD1. Animal models deficient in PD1 signaling are prone to autoimmune diseases similar to SLE. PD-L1 and PD1 expression on blood cells is low in SLE patients, and polymorphisms in the gene for PD1 are associated with SLE. PD-L1 is highly expressed by healthy placental trophoblasts, where it blocks the activation and cytokine production by alloreactive maternal T lymphocytes to protect the fetus.

Little is known about placental costimulatory molecules in women with autoimmune diseases; preliminary data suggest decreased overall PD-L1 expression in SLE placentas.

The long-term goal of this project is to improve clinical practice by identifying novel PD1-binding peptide moieties that represent biologic therapy used in utero and in neonates to improve maternal-fetal tolerance and prevent autoimmunity in the mother and her progeny.

## Institution

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