"The lupus patient has a normal response to an unrelated donor. And [the patient has] twice that response to her mother. So she's hyperactivated," said Dr. Stevens, a researcher at the Children's Hospital and Regional Medical Center, Seattle.

"Somehow the control patients had developed an immune tolerance to maternal antigens. Only 2% of their cells are actually responding to the mother's, which would explain why they can tolerate maternal microchimerism for decades."

These results are still preliminary, Dr. Stevens commented. "This has to be substantiated in many, many more people."

However, if additional evidence shores up her hypothesis, and maternal cells are shown to trigger an autoimmune response in SLE patients, clinicians might be able to halt the disease.

"Conceivably, we could target these maternal antigens or block the maternal HLA molecules and specifically stop this immune response. There are such a tiny number of cells involved that you would not be wiping out huge amounts of tissue. You'd be getting rid of the stimulus, and the rest of the body would be fine," Dr. Stevens said. ■

More Antibodies Implicated in SLE Nephritis

BY ROBERT FINN
San Francisco Bureau

Individuals with systemic lupus erythematosus who go on to develop nephritis are more than five times as likely to have antibodies to lipoprotein lipase in their blood serum, according to findings from an investigation led by Morris Reichlin, M.D.

This suggests that the pathogenesis of lupus nephritis may involve cell-surface antigens that activate the complement system and promote vascular damage in the kidney and other organs when they are engaged by antibodies.

Other antibodies have previously been shown to be associated with systemic lupus nephritis, wrote Dr. Reichlin of the University of Oklahoma, in Oklahoma City.

These antibodies include anti-double-stranded DNA (anti-dsDNA), anti-ribosomal P protein (anti-P), anti-Ro/SSA, antihistones, anti-C_1q, and antinucleosomes. This is the first study to demonstrate an association between lupus nephritis and anti-lipoprotein lipase (anti-LPL).

According to the study, anti-LPL shows strong relationship to this SLE complication than any single specificity (Clin. Immunol. 2005;117:12-4).

In addition, Dr. Reichlin found that SLE patients with anti-LPL and anti-P antibodies were more than 17 times more likely to develop lupus nephritis than were those who had neither antibody. This result was highly significant, with a *P* value of .00002.

The study involved 35 patients with SLE who had developed nephritis that apparently had no other cause and 28 patients with SLE who had no evidence of nephritis.

Twenty-five (71.4%) of the patients with nephritis had anti-LPL antibodies in their serum compared with 9 (32%) of the patients without nephritis.

Twenty (57.1%) of the patients with nephritis had both anti-LPL and anti-P, compared with just two (7.1%) of the patients who as yet have no clinical evidence of nephritis.

"It will be of interest to follow those two patients to assess their outcomes," Dr. Reichlin wrote. ■