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Abstract

# Gut microbiota dysbiosis and altered tryptophancatabolism contribute to autoimmunity in lupus-susceptible mice

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## Abstract:

The autoimmune disease systemic lupus erythematosus (SLE) is characterized by the production of pathogenic auto- antibodies. It has been postulated that gut microbial dysbiosis may be one of the mechanisms involved in SLE pathogenesis. Here, we demonstrate that the dysbiotic gut microbiota of triple congenic (TC) lupus-prone mice (B6.Sle1.Sle2.Sle3) stimulated the production of autoantibodies and activated immune cells when transferred into germfree congenic C57BL/6 (B6) mice. Fecal transfer to B6 mice induced autoimmune phenotypes only when the TC donor mice exhibited autoimmunity.

## Biography:

Foreign Research Visitor, Department of Entomology, University of Georgia, 1985-1986. Post-doctoral Assistant, Department of Pathology and Laboratory Medicine, College of Medicine, University of Florida, 1991-1992. Visiting Assistant IN, The postdoctoral program of the Department of Pathology and Laboratory Medicine, College of Medicine, University of Florida, 1993-1996. Assistant Scientist, Department of Pathology and Laboratory Medicine, College of Medicine, University of Florida, 1996-1998. Member of the Center for Mammalian Genetics, 1996-2005. Assistant Professor, Departments of Medicine, and Pathology, Immunology, and Laboratory Medicine University of Florida, January 1999-2004.


## Recent Publications:

1. Intestinal Dysbiosis and Tryptophan Metabolism in Autoimmunity
2. Gut microbiota dysbiosis and altered tryptophan catabolism contribute to autoimmunity in lupus-susceptible mice
3. T cells expressing the lupus susceptibility allele Pbx1d enhance autoimmunity and atherosclerosis in dyslipidemic mice
4. Efficacy of the Combination of Metformin and CTLA4Ig in the (NZB × NZW)F1 Mouse Model of Lupus Nephritis

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