



Illinois Wesleyan University
Digital Commons @ IWU

John Wesley Powell Student Research
Conference

2008, 19th Annual JWP Conference

Apr 12th, 9:00 AM - 10:00 AM

Functional Analysis of the Human FCGRT Promoter Polymorphisms

Brian Rea
Illinois Wesleyan University

Dr. Robert Kimberly, Faculty Advisor
Illinois Wesleyan University

Dr. Kaihong Su, Faculty Advisor
University of Alabama at Birmingham

Follow this and additional works at: <https://digitalcommons.iwu.edu/jwprc>

Rea, Brian; Kimberly, Faculty Advisor, Dr. Robert; and Su, Faculty Advisor, Dr. Kaihong, "Functional Analysis of the Human FCGRT Promoter Polymorphisms" (2008). *John Wesley Powell Student Research Conference*. 20.
<https://digitalcommons.iwu.edu/jwprc/2008/posters/20>

This Event is protected by copyright and/or related rights. It has been brought to you by Digital Commons @ IWU with permission from the rights-holder(s). You are free to use this material in any way that is permitted by the copyright and related rights legislation that applies to your use. For other uses you need to obtain permission from the rights-holder(s) directly, unless additional rights are indicated by a Creative Commons license in the record and/ or on the work itself. This material has been accepted for inclusion by faculty at Illinois Wesleyan University. For more information, please contact digitalcommons@iwu.edu.

©Copyright is owned by the author of this document.

Poster Presentation P41

**FUNCTIONAL ANALYSIS OF THE HUMAN FCGRT PROMOTER
POLYMORPHISMS**

Brian Rea and Dr. Robert Kimberly* and Dr. Kaihong Su*
Biology Department, Illinois Wesleyan University
University of Alabama at Birmingham

Recent studies have indicated that the neonatal Fc Receptor may play a role in autoimmune diseases such as Systemic Lupus Erythematosus (SLE). The neonatal Fc receptor (FCGRT, also called FcRn) mediates transmission of immunoglobulin G (IgG) from mother to fetus perinatally and also plays a central role in the protection of serum IgG from catabolism. The expression levels of FCGRT directly correlate with serum IgG concentrations. FCGRT deficient mice were protected against SLE because serum IgG level is not at the concentration necessary to produce autoantibodies leading to the disease. Therefore, understanding the transcriptional regulation of the human FCGRT gene and how genetic polymorphisms in the promoter affect the receptor expression will help identify alleles that contribute to autoimmune diseases. In this study, we sequenced 20 individuals and identified a 37 base pair deletion in the promoter region. Promoter reporter analyses in both monocytic U937 cells and epithelial MDA468 cells demonstrated that the proximal 1 kb promoter of human FCGRT confers the greatest promoter activity and the 1 kb promoter with the 37 nucleotide deletion leads to decreased promoter activity. Our data suggests that the transcriptional control of the human FCGRT gene may reside within 1 kb proximal promoter and the deletion polymorphism decreases the promoter activity. These results provide insights for the regulation of the endogenous FCGRT receptor expression and predict that individuals with the deletion polymorphism express lower levels of FCGRT. Lower levels of FCGRT may indicate that the deletion polymorphism acts as a protection against autoimmune disease.