



**CALIFORNIA STATE SCIENCE FAIR
2003 PROJECT SUMMARY**

Name(s) Khang D. Nguyen	Project Number S0416
Project Title Mutations in Nod2 Affect NF-kB Activation	
Abstract Objectives/Goals The purpose of this project is to determine if mutations in Nod2, a gene thought to be involved in Crohn's disease, affects the activation of the inflammatory response due to induction of NF-kB. Methods/Materials Dr. Laurie Bankston made two deletion mutations in the mouse gene Nod2. I transfected these mutations along with control constructs into 293 cells (human embryo fibroblast continuously maintained cell line). The total protein of the 293 cells was extracted and a luciferase assay was done to determine the amount of NF-kB activation. The relative amounts that each mutation activated NF-kB was compared. Results Overexpression of wild type (N2F) and the two mutations (N2M, N2M2) caused activation of NF-kB. Wild type and N2M activated NF-kB to about the same level while the N2M2 induced higher activation. The amount of activation by N2M2 was about three-quarters of IKKbEE (constitutively active, positive control). These arbitrary numerical values were compared to the b-actin (empty vector), the negative control. Conclusions/Discussion A high percentage of patients with Crohn's disease have the N2M mutation. It is not certain whether this mutation is really the cause of the disease. From the results of this project, we can conclude that N2M activates NF-kB at least as well as wild type in overexpression experiments. Thus, it is a potential candidate for causing Crohn's disease.	
Summary Statement To determine if mutations in Nod2, a gene thought to be involved in Crohn's disease, affects the activation of the inflammatory response due to induction of NF-kB.	
Help Received Used lab equipment at the Basic Science Building on the campus of the University of California, San Diego, under the supervision of Greg Cadwell, Staff Research Associate III	