Raju Kucherlapati, Ph.D.

Department of Molecular Genetics
Albert Einstein College of Medicine
1300 Morris Park Avenue
Bronx, New York 10461
USA

Dr. Kucherlapati is the Lola and Saul Kramer Professor and Chairman of the Department of Molecular Genetics at Albert Einstein College of Medicine in Bronx, New York. He received his Ph.D. degree in Genetics from the University of Illinois. After conducting post-doctoral work at Yale University, he joined the faculty at Princeton University. Dr. Kucherlapati was a Professor of Genetics at the University of Illinois College of Medicine prior to joining the faculty of the Albert Einstein College of Medicine. Dr. Kucherlapati's current research interests include structural and functional genomics and the development of mouse models for human disease.

Role of colon cancer genes in development

Mutations in the adenomatous polyposis coli gene (APC) result in familial adenomatous polyposis (FAP), an autosomal dominant disorder that is marked by susceptibility to gastrointestinal tumours. The product of APC participates in maintaining the cytoplasmic levels of β-catenin. APC gene mutations lead to accumulation of cytoplasmic β-catenin, which translocates to the nucleus, binds to TCF4 and activates transcription of a number of important target genes. To understand the role of APC, we developed mice that carry a mutation in Apc. The mutation is designated Apc1638N. Mice that are heterozygous for Apc1638N exhibit a tumour susceptibility phenotype. This mutation causes embryonic lethality in the homozygous state. To understand the role of Apc in development, we developed homozygous Apc1638N embryonic stem (ES) cell lines. We examined the patterns of gene expression in these cells and compared them with those in wild-type ES cells. cDNA microarrays were prepared by a robotic device built at Einstein and analysed by a scanner also built at Einstein. Analysis of data with publicly available tools as well as those we developed allowed us to identify a number of genes whose expression is modulated in Apc-deficient cells.