

Mammalian Biology: Immunology

**Group Leader**

## **Kanury Venkata Subba Rao**

ICGEB Laboratories  
ICGEB Campus  
Aruna Asaf Ali Marg  
110 067 New Delhi, INDIA



E-mail: [kanury@icgeb.res.in](mailto:kanury@icgeb.res.in)  
Office tel: +91-11-26741680  
Office fax: +91-11-26742316

## **Education**

Pune University, Department of Chemistry, Pune, India, MSc, 1979  
MS University, Department of Chemistry, India, PhD, 1983

## **Career History**

Since 1995, Group Leader of the Immunology Group, International Centre for Genetic Engineering and Biotechnology (ICGEB), New Delhi, India.  
1991-1994, Assistant Scientist, ICGEB New Delhi.  
1988-1991, Research Scientist, ICGEB New Delhi.

## **Teaching Activity**

Tutoring activities in the ICGEB Ph.D. Programme.

## **Scientific Activity**

Our long-term interest is in understanding the regulatory parameters that influence the outcome of an immune response. Within this our current focus is to understand the mechanisms controlling plasticity in receptor-initiated signaling pathways. The experimental strategy involves a 'systems level' analysis of the antigen receptor-dependent signal transduction network. We measure the kinetic and quantitative contributions of various intermediates in the signaling pathways and then monitor how these contributions are modulated in response to specific perturbations within the network. Our larger goal is to delineate the overall structure of this network and also to characterize its dynamic features. We are particularly interested in deriving both quantitative and qualitative measures of signal output under conditions where the receptor is differentially activated. The aim here is to eventually understand how such information processing impacts on the gene expression profile.

## **Selected publications**

Sharma, M., George, A.A., Singh, B.N., Sahoo, N.C., Rao, K.V.S. 2007. Regulation of Transcript Elongation through Cooperative and Ordered Recruitment of Cofactors. *J. Biol. Chem.* 282, 20887-20896

Basu, S.K., Kumar, D., Singh, D.K., Ganguly, N., Siddiqui, Z., Rao, K.V.S., and Sharma, Pawan. 2006. Mycobacterium tuberculosis secreted antigen (MTSA-10) modulates macrophage function by redox regulation of phosphatases. *FEBS J.* 273, 5517-5534

George, A.A. Sharma, M., Singh, B.N., Sahoo, N.C., Rao, K.V.S. 2006. Transcription from a TATA and INR-less promoter: Spatial segregation of promoter function. *EMBO J.* 25, 811-821

Sethi, D.K., Agarwal, A, Manivel, V., Rao, K.V.S., Salunke, D.M. 2006. Differential epitope positioning within the germline antibody paratope enhances promiscuity in the primary immune response. *Immunity* 24, 429-438

Sinha A, A Singh, V Satchidanandam and K Natarajan. 2006. Impaired generation of reactive oxygen species during differentiation of Dendritic cells (DCs) by Mycobacterium tuberculosis secretory antigen (MTSA) and subsequent activation of MTSA-DCs by mycobacteria results in increased intracellular survival. *J Immunol.* 177, 468-478

Latchumanan, V., Balkhi, M.Y., Sinha, A., Singh, B., Sharma, P., Natarajan, K. 2005. Regulation of immune responses to Mycobacterium tuberculosis secretory antigens by Dendritic cells. *Tuberculosis* 85, 377-383

Singh, D.K., Kumar, D., Siddiqui, Z., Basu, S.K., Kumar, V., Rao, K.V.S. 2005. The strength of receptor signaling is centrally controlled through a cooperative loop between Ca<sup>2+</sup> and an oxidant signal. *Cell* 121, 281-293

Chaturvedi, A., Siddiqui, Z., Bayiroglu, F., Rao, K.V.S. 2002. A GPI-linked isoform of the IgD receptor regulates resting B cell activation. *Nature Immunol.* 3, 951-957

Manivel, V., Bayiroglu, F., Siddiqui, Z., Salunke, D.M., Rao, K.V.S. 2002. The primary antibody repertoire represents a linked network of degenerate antigen specificities. *J. Immunol.* 169, 888-897

Natarajan, K., Sahoo, N.C., Rao, K.V.S. 2001. Signal thresholds and modular synergy during expression of costimulatory molecules in B lymphocytes. *J. Immunol.* 167, 114-122

Manivel, V., Sahoo, N.C., Salunke, D.M., Rao, K.V.S. 2000. Maturation of a primary antibody response is governed by modulations in flexibility of the antigen-combining site. *Immunity* 13, 611-620