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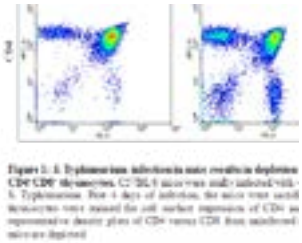
Investigations on sub-populations of thymocytes during atrophy

T cells are critical for cell-mediated immunity and these cells are generated in the thymus. The thymus is a primary lymphoid organ in which T cells differentiate and mature. Upon stringent selection, only mature T cells egress into the periphery and are responsible for cellular immunity. There are four major sub-populations of thymocytes based on expression of CD4 and CD8 markers: $CD4^-$ $CD8^-$ or double negative, $CD4^+$ $CD8^+$ or double positive, $CD4^+$ single positive and $CD8^+$ single positive cells. Thymic atrophy, *i.e.* the loss in the number of thymocytes, occurs during aging, stress and infections. Indeed, thymic atrophy is also observed upon infection with viruses, bacteria, fungi etc.; however, the reasons and consequences of thymic atrophy are not well understood. To better understand the molecular and cellular processes involved during thymic atrophy, we standardized a model of infection-induced thymic atrophy using the intracellular bacterial pathogen, *Salmonella typhimurium*. During oral infection of C57BL/6 mice by *Salmonella typhimurium* the number of immature $CD4^-$ $CD8^-$ and $CD4^+$ $CD8^+$ thymocytes, but not single positive and mature $CD4^+$ or $CD8^+$ thymocytes or mesenteric lymph node cells, were greatly reduced. In this presentation, I will outline some of our recent work regarding the subsets that are affected during thymic atrophy during *Salmonella typhimurium* infection. In addition, we are studying thymic sob-populations in other modes of atrophy, *i.e.* caused by lipopolysaccharide (LPS), etoposide which is an anti-cancer compound and dexamethasone, a synthetic glucocorticoid. Preliminary data on differences in thymic sub-populations in different modes of thymic atrophy and possible mechanistic insights will be presented. Better understanding of the processes involved in thymic atrophy may lead to the development strategies that may boost the cellular response during stress, infections, treatments with anti-cancer drugs etc.

Recent publications

1. Majumdar S, Deobagkar Lele M, Adiga V, Raghavan A, Wadhwa N et al. (2017) Differential susceptibility and maturation of thymocyte subsets during *Salmonella typhimurium* infection: Insights on the roles of glucocorticoids and interferon-gamma. Scientific Reports. 7: 40793
2. Podder S, Rakshit S, Ponnusamy M, Nandi D (2016) Efficacy of bacteria in cancer immunotherapy: Special emphasis on the potential of mycobacterial Species. Clinical Cancer Drugs. 3(2):100-108.
3. Deobagkar Lele M, Victor E S, Nandi D (2014) c-Jun N-terminal Kinase is a critical node in the death of CD4+ CD8+ thymocytes during *Salmonella enterica* serovar *typhimurium* infection. European Journal of Immunology. 44(1): 137-149.
4. Deobagkar Lele M, Chacko S, Victor E S, Kadthur J C, Nandi D (2013) Interferon-γ and glucocorticoid mediated pathways synergize to enhance death of CD4+ CD8+ thymocytes during *Salmonella enterica* serovar *typhimurium* infection. Immunology. 138(4):307-321.
5. Bhosale M, Kadthur J C, Nandi D (2012) Roles of *Salmonella enterica* serovar *typhimurium* encoded Peptidase N during systemic infection of *Ifn* $\gamma^{-/-}$ mice. Immunobiology. 217(3): 354-362.



Biography

Dipankar Nandi is a Professor in the Department of Biochemistry, IISc, Bangalore, India. He performed his Doctoral studies at the University of California, Berkeley, USA on characterization of epithelial tissue associated T cells and was awarded the PhD degree in 1991. He was introduced to the world of protein degradation during his Postdoctoral Research at the University of Cincinnati, Ohio, USA while studying the composition and assembly of Interferon-γ-inducible proteasomes. He joined IISc in 1997 and has developed a group with broad research interests: microbial responses to stress, mechanisms involved in antibiotic resistance etc. and host responses to cytokines, infection etc. He has published more than 60 scientific papers and has supervised 12 students for PhD. He is a reviewer for more than 10 international journals.

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