

## **Santo Landolfo, University of Turin, Italy**

Santo Landolfo is a Full Professor of Microbiology at the Head Laboratory of Molecular Virology, Department of Public Health and Microbiology, Medical School at the University of Turin in Italy. He received his Medical degree at the University of Turin, Italy in 1974, and his Ph.D. in Microbiology in 1978.

- 1975-1978: Visiting Fellow, National Cancer Institute, NIH, Bethesda, with a project addressed to study the role of cytokines in the immunity against tumors induced by murine retroviruses (MSV, MLV).
- 1978-1980: Fellowship of the Italian National Research Council (CNR) to continue the project on the immunity against tumors induced by MSV.
- 1980-1982: Visiting Associate at the German National Research Institute, DKFZ, Heidelberg, with a research program addressed to explore the mechanisms of IFN- production by cells of the immune system and the mechanism responsible for IFN-antiproliferative activity.
- 1982-1985: Assistant Professor, Medical School, Turin. During this period the proponent defined the molecular structure of IFN- $\gamma$  and demonstrated that IFN- $\gamma$  is one of the main mediators of allograft rejection.
- 1985-2009: Full Professor of Microbiology, Medical School of Turin.
- 1997-2002: Director of the Immunogenetic and Experimental Oncology Center, CNR, Turin.

Main research interests:

A) Characterization of the human and mouse IFN-inducible HIN200 gene family. Overexpression of IFI16, a member of the HIN200 family, in primary human endothelial cells leads to inhibition of tube morphogenesis accompanied by increased expression of pro-inflammatory molecules. Thereafter cells undergo apoptosis with apoptotic bodies displaying IFI16 on their surface. Consistent with these results, the presence of autoantibodies against IFI16 has been observed in the sera from patients affected by autoimmune diseases, including Systemic Scleroderma (SSc), Systemic Lupus Erythematosus (SLE) and Sjogren Syndrome (SjS), suggesting a role of IFI16 protein in the etiopathogenesis of the above autoimmune diseases.

B) The role of human cutaneous Papillomaviruses (HPV) in skin carcinogenesis. In collaboration with the Laboratory of Virology, Novara, University of Piemonte Orientale (UPO) we have been studying the mechanisms of epithelial transformation by cutaneous HPV. The outcome of these studies demonstrate that, whereas mucosal HPV immortalize human keratinocytes by modulating the activity of a complex of proteins regulating the cell cycle and proliferation (p53, pRb, human telomerase etc.), tumor progression triggered by cutaneous HPV genotypes appears to depend on the microenvironment influenced by the inflammatory response of the host.