# Umberto Dianzani

*Curriculum vitae*

## PERSONAL DATA

Born at Genoa, June 21th, 1959
Resident at Torino
Mobile phone: 3498724605

## BIO AND EDUCATION

-1984.: MD degree, University of Turin, Italy (cum laude)
-1985. Post-doc, Immunocytology laboratory, Department of Pathology, University of Alabama at Birmingham, AL.
-1988-89. Posgt-doc Section of Immunobiology, Yale University, CT.
-1990. PhD degree in Experimental Hematology, University of Genoa, Italy

Member of the Italian Society of Immunology Clinical Immunology and Allergy, the Italian Society of Pathology and Translational Medicine, the American Association of Immunologists, and the Medical Academy of Turin.
Coauthor of 150 scientific publications. Total impact factor >750, H Index 35, total citations >4000

Main research achievements:
1-Costimulatory activity of CD73 in T cells
2-Lateral association of surface receptors in T cells
3-Adhesive activity of CD38 and CD31
4-H4/ICOS, a novel costimulatory molecule of T cells
5-Role of Fas, perforin and osteopontin in autoimmune diseases
6-Role of ICOSL in several cell types

## UNIVERSITY CAREER

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| 1999-2016 | Full professor ordinario, Università del Piemonte Orientale |
| 1998-1999 | Associate professor, Università del Piemonte Orientale |
| 1993-1998 | Associate professor, Università di Torino, sede di Novara |
| 1992-1993 | Associate professor, Università di Pisa |
| 1990-1992 | Research assistant, Università di Torino |

## UNIVERSITY POSITIONS

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| 2012-2016 | Academic Senate member, Università del Piemonte Orientale |
| 2012-2016 | Head of Department of Health Sciences, Università del Piemonte Orientale |

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| 2011-2015 | Delegate of the Rector for International Relationships |
| 2010-2011 | Vice-dean of the Faculty of Medicine, Università del Piemonte Orientale |
| 2000-2012 | Coordinator of the PhD program in Molecular Medicine |

## SCIENTIFIC POSITIONS

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| 2002-2010 | President of the Interdisciplinary Research Centor for Autoimmune Diasease (IRCAD), Novara. |

## MAIN FIELDS OF INTEREST

1. Immunology
2. T cell costimulatory receptors
3. Pathogenetic mechanisms of autoimmune diseases
4. Apoptosis

## CURRENT ISSUES OF RESEARCH

### 1. Role of ICOS/ICOSL in the immune response

ICOS is expressed by T cells and binds ICOSL expressed by several immune and not immune cell types. ICOS triggering modulates cytokine secretion in T helper cells, whereas triggering of ICOSL modules function of dendritic cells and adhesion/migration of several cell types. The ongoing research analyzes novel mechanisms of the ICOS and ICOSL activity on several cell types, including bone cells and tumor cells, and looks for pharmacological modulators of these activities.

### 2. Role of osteopontin in the immune response

Osteopontin (OPN) is involved in bone mineralization, but it i also a proinflammatory cytokine with chemotactic activity and capable to influence the T helper response. Our ongoing research in investigating modulation of OPN activity by several types of proteases, looking for the peptides produced upon cleavage, their function and receptors, and the role in autoimmune and neoplastic disease.

### 3. Development of tolerogenic vaccines for autoimmune diseases

Tolerogenic vaccines for autoimmune diseases are aimed to suppress the autoimmune response with an (auto)antigen-specific approach. Our ongoing research is developing a novel generation of tolerogenic vaccines by loading autoantigens and immunomodulators in biocompatible and biodegradable nano/microparticles.

### 4. Analysis of apoptosis alterations in autoimmune diseases

Functional alterations of apoptos play a role in development of several autoimmune diseases and involve molecules such as Fas, FasL, perforin, osteopontin and IL-17. Our ongoing research is investigating the role of other molecules which may play a role in these defects, such as TNFSF14, TNFRSF14 and PIM-1.

## CURRENT FUNDED PROJECTS

| BANDO | TITOLO DEL PROGETTO |
|---|---|
| AIRC | ROLE OF ICOS/B7H IN THE ANTI-TUMOR IMMUNE RESPONSE. |
| FISM | GENOMIC AND FUNTIONAL STUDY OF THE ROLE OF THE NFSF14/TNFRSF14 PATHWAY IN MULTIPLE SCLEROSIS SUSCEPTIBILITY |

## TOP FIVE PAPERS


1. Dianzani U, Luqman M, Rojo J, Yagi J, Baron J L, Woods A, Janeway C A and Bottomly K.: Molecular associations on the T cell surface correlate with immunological memory. *European Journal Immunology* 20: 2249-2257, 1990.
2. U. Dianzani, M. Bragardo, D. DiFranco, C. Alliaudi, P. Scagni, D. Buonfiglio, V. Redoglia, S. Bonissoni, A. Correra, I. Dianzani, and U. Ramenghi. Deficiency of the Fas apoptosis pathway without Fas gene mutations in pediatric patients with autoimmunity/lymphoproliferation. *Blood* 89:2871-2879, 1997.
3. Nurieva RI, Duong J, Kishikawa H, Dianzani U, Rojo JM, Ho I, Flavell RA, Dong C. Transcriptional regulation of Th2 differentiation by inducible costimulator. *Immunity*. 18:801-811, 2003.
4. Clementi R, Dagna L, Dianzani U, Dupré L, Dianzani I, Ponzoni M, Cometa A, Chiocchetti A, Sabbadini MG, Rugarli C, Ciceri F, Maccario R, Locatelli F, Danesino C, Ferrarini M, Bregni, M. Inherited Perforin and *Fas* Mutations in a Patient with Autoimmune Lymphoproliferative Sindrome and Lymphoma. *N Engl J Med 351*:1419-24, 2004.
5. Chiocchetti A, Indelicato M, Bensi T, Mesturini R, Giordano M, Sametti S, Castelli L, Bottarel F, Mazzarino MC, Garbarini L, Giacopelli F, Valesini G, Santoro C, Dianzani I, Ramenghi U, Dianzani U. High levels of osteopontin associated with polymorphisms in its gene are a risk factor for development of autoimmunity/lymphoproliferation. *Blood* 103:1376-82, 2004.