

## Curriculum Vitae

### Walter Malorni, Ph.D.

**Current address:** Department of Therapeutic Research and Medicines Evaluation, Italian National Institute of Health, Viale Regina Elena 199, 00161 Rome, Italy - E-mail: walter.malorni@iss.it

After his Biological Sciences Degree in 1976 in the Laboratory of Psychopharmacology carried out with the Nobel price Daniel Bovet, from 1978 to 1983 he was Fellow at the Regina Elena Institute for Cancer Research, Rome, Italy. From 1984 to 1988 he was Researcher in the Department of Ultrastructures, Istituto Superiore di Sanità, in Rome, where from 1988 he was the Head of the Section of Subcellular Pathology. Nowadays he is Head of the Section "Cell aging and degeneration" at the Department of Drug Research and Evaluation at Istituto Superiore di Sanità (Italian National Institute of Health).

Since 1998 Dr. Malorni was also professor at the University of Modena and of L'Aquila teaching "Cellular toxicology" and "Clinical biochemistry", and at the University of L'Havana (Cuba) and San Paulo (Brazil) teaching cell pathology.

He is reviewer for more than 20 international journals for Italian and international projects. He is author of more than 220 articles included in SCI.

Granted from Telethon, National Research Council, Italian Ministry of Research, Italian Ministry of Health, European Community, National Institute of Health (USA). His main interests are focused on cell degeneration and apoptosis in cancer.

#### Selected publications:

- 1: Matarrese P, Straface E, Palumbo G, Anselmi M, Gambardella L, Ascione B, Del Principe D, Malorni W. Mitochondria regulate platelet metamorphosis induced by opsonized zymosan A - activation and long-term commitment to cell death. *FEBS J.* 2009 Feb;276(3):845-56.
- 2: Maselli A, Matarrese P, Straface E, Canu S, Franconi F, Malorni W. Cell sex: a new look at cell fate studies. *FASEB J.* 2008 Dec 12.
- 3: Degli Esposti M, Tour J, Ouasti S, Ivanova S, Matarrese P, Malorni W, Khosravi-Far R. Fas death receptor enhances endocytic membrane traffic converging into the Golgi region. *Mol Biol Cell.* 2009 Feb;20(2):600-15.
- 4: Ortona E, Margutti P, Matarrese P, Franconi F, Malorni W. Redox state, cell death and autoimmune diseases: a gender perspective. *Autoimmun Rev.* 2008 Jul;7(7):579-84.
- 5: Matarrese P, Manganelli V, Garofalo T, Tinari A, Gambardella L, Ndebele K, Khosravi-Far R, Sorice M, Esposti MD, Malorni W. Endosomal compartment contributes to the propagation of CD95/Fas-mediated signals in type II cells. *Biochem J.* 2008 Aug 1;413(3):467-78.
- 6: Malorni W, Straface E, Matarrese P, Ascione B, Coinu R, Canu S, Galluzzo P, Marino M, Franconi F. Redox state and gender differences in vascular smooth muscle cells. *FEBS Lett.* 2008 Mar 5;582(5):635-42.
- 7: Franconi F, Seghieri G, Canu S, Straface E, Campesi I, Malorni W. Are the available experimental models of type 2 diabetes appropriate for a gender perspective? *Pharmacol Res.* 2008 Jan;57(1):6-18.
- 8: Minetti M, Leto TL, Malorni W. Radical generation and alterations of erythrocyte integrity as bioindicators of diagnostic or prognostic value in COPD? *Antioxid Redox Signal.* 2008 Apr;10(4):829-36.
- 9: Margutti P, Matarrese P, Conti F, Colasanti T, Delunardo F, Capozzi A, Garofalo T, Profumo E, Riganò R, Siracusano A, Alessandri C, Salvati B, Valesini G, Malorni W, Sorice M, Ortona E. Autoantibodies to the C-terminal subunit of RLIP76 induce oxidative stress and endothelial cell apoptosis in immune-mediated vascular diseases and atherosclerosis. *Blood.* 2008 May 1;111(9):4559-70.
- 10: Minetti M, Agati L, Malorni W. The microenvironment can shift erythrocytes from a friendly to a harmful behavior: pathogenetic implications for vascular diseases. *Cardiovasc Res.* 2007 Jul 1;75(1):21-8.



*Scientific Session on:*  
**INTEGRATION OF GENDER MEDICINE  
IN THE CLINICAL PRACTICE**  
Saturday, November 7<sup>th</sup>, 2009  
Berlin – Germany

**CELL SEX: A NEW LOOK AT CELL  
FATE STUDIES**

Elisabetta Straface, Paola Matarrese  
and **Walter Malorni**  
Italian National Institute of Health,  
Department of Therapeutic Research  
and Medicines Evaluation



Different pathways involved in the complex machinery implicated in determining cell fate have been investigated in the recent years. Different forms of cell death have been described: apart from the "classical" form of death known as necrosis, a well characterized traumatic injury of the cell, several additional forms of cell death have been identified. Among these, apoptosis has been characterized in detail. These studies stem from the implication that the apoptotic process plays a key role in human pathology. In fact, defects in the mechanisms of cell death, i.e. both an increase or a decrease of apoptosis, have been associated with the pathogenesis of a number of human diseases. Some new insights also derive from the study of autophagy, a less characterized form of cell damage mainly associated with cell survival strategies but that also leads, as final event, to the death of the cell. Interestingly, very recently, a gender difference has been found in this respect: cells from males and females can behave differently. In fact, they seem to display several different features (reactive oxygen species formation, cytoskeleton assembly etc), including those determining their fate. The idea that primary cultured cells or ex vivo cells could maintain their sexual features can disclose new scenarios in preclinical and clinical studies in the field of both disease pathogenesis and pharmacology.