Prof. Antonella Forlino

1991-University of Pavia-Degree in Biological Science;
1994- University of Pavia-Doctoral Degree (PhD) in Biochemistry;
1997- University of Pavia -Speciality School in Genetic;
1995-1999- National Institute of Health, Bethesda, Maryland, USA-Research Fellow;
2001-2010 Department of Biochemistry, University of Pavia-Researcher;
2010- Department of Molecular Medicine, Biochemistry Unit, University of Pavia, Associate Professor

Teaching activities

Professor of Biochemistry in the Harvey Course, Faculty of Medicine, University of Pavia
Professor of Biochemistry in the Golgi Course, Faculty of Medicine, University of Pavia
Professor of Biochemistry in the Biotechnology Master Degree

Prof. Forlino's research activity is on the molecular, biochemical, and functional study of genetic diseases of the connective tissue, in particularly Osteogenesis Imperfecta (OI), and Prolidase Deficiency (PD).

Classic Osteogenesis Imperfecta is a bone disease caused by mutations in the two genes coding for the α chains of Type I collagen. Dr Forlino's research is focused on the investigation of the mutated collagens produced by cultured fibroblasts and osteoblasts obtained from OI patients. She studies the effects of the mutated collagens on the intracellular homeostasis and on other extracellular matrix proteins.
She characterized the molecular defect in some OI patients contributing to the hypothesis of a regional model to explain the genotype/phenotype relationship in OI.
During her fellowship in USA she generated and characterized the first knock-in murine model of OI (BrtlIV). She is actually using the model together with another murine model of dominant OI to better understand the phenotypic variability characteristic of OI using microarray techniques and proteomic approach.
She developed for the OI murine model BrtlIV an in utero cell therapy approach using whole bone marrow obtained by GFP transgenic mice and she is now involved in a gene therapy project focused on targeting the expression of the mutant Brtl allele.
Prof. Forlino set up a zebrafish facility in Pavia to generate by CRISPR/Cas9 genome editing OI zebrafish models for dominant and recessive OI in order to attempt drug screening analysis aimed to identify novel treatments for the disease.
She actively collaborates with her former laboratory at the National Institute of Health, Bethesda, MD, USA (Dr. Marini's Laboratory).

Prolidase Deficiency is a skin disease caused by mutations in the gene coding for prolidase, a metallo dipeptidase responsible for the hydrolysis of dipeptides containing Proline or Hydroxyproline at the C-terminal end. Dr Forlino's research on Prolidase Deficiecy was focused on the characterization of new mutations in PD patients and on the development of a new hypothesis to explain the molecular bases of some clinical symptoms in PD patients.
She generated two expression systems, eukariotic and prokaryotic respectively, to obtain human recombinant prolidase to develop an enzyme replacement therapy for prolidase deficiency.

She characterized the bone defect in the knock in murine model of PD in collaboration with Dr Phang, NCI, USA.

She obtained as principal investigator some national and international grants (Telethon 2015, Telethon 2013, Cariplo 2013, AFM 2012, PRIN2006: 2006050235; Fondazione Cariplo, 2007; Progetto Giovani Ricercatori, 2000 and American OI Foundation, 2001/2002).

She is member of the European Calcified Tissue Society, of the Italian Society of Connective Tissue, Italian Association of O.I. (AS.IT.O.I.) and of the Italian Stem Cell Society.

She is author of over 75 peer reviewed paper in international journals.