Università di Genova

Roberta De Tullio

Associate Professor

EDUCATION AND TRAINING

1993: One year Research fellowship awarded by the National Research Council (CNR) (P.R. Genetics)
1992: One year Research fellowship awarded by the National Research Council (CNR) (P.R. Biochemistry)
1992: Ph.D. Biochemistry Universities of Genova and Pavia, Italy
1988: Biologist qualifying examination
1983-1987: Degree in Biological Sciences, University of Genova, Italy
1979-1983: High school diploma in Languages, G. Deledda, Genova, Italy

PROFESSIONAL HISTORY

2022-present: Associate Professor BIO/10 Biochemistry, at Department of Experimental Medicine (DIMES), University of Genova, Italy
2017: National Academic Qualification (ASN) as Associate Professor in General Biochemistry (05/E1)
1995-2022: Researcher Ph.D. at Department of Experimental Medicine (DIMES), University of Genova,

ACADEMIC APPOINTMENTS

2022-present: Member of the teaching board of the Dietistic school, University of Genova,
2013-present: Member of the teaching board of the Dentistry school, University of Genova, Italy
2018-2023: Member of Joint Teachers-Students Commission for teaching and the right to study, University of Genova, Italy
2004-2014: Member of the Ph.D. board in Biochemistry University of Genova
2001-2020: Member of the Center for Excellence in Biomedical Research (CEBR), University of Genova
1995-present: Member of the Italian Society of Biochemistry and Molecular Biology (SIB)

EXPERIENCE

- GRANTS 2022-2024: PI of Operative Unit Genova PRIN 2022 (Programmi di Ricerca Scientifica di Rilevante Interesse Nazionale) (24 months, Prot. 2022JBR44Z) (PI Coordinator Paola Costelli University of Torino) “The energy crisis in the nerve-muscle system as a driver of cancer cachexia: innovative mitochondria-targeted strategies.”

- REVIEWER ACTIVITY for international peer-review journals including Biochimica et Biophysica Acta (BBA), iScience, Journal of Hematology and Blood Disorders (JHBD), AIMS Molecular Science and others

-TEACHING ACTIVITY Associate Professor in Biochemistry of the School in Medical Pharmaceutical Sciences, University of Genova:
-Chemistry and Propaedeutic Biochemistry: School of Dental Medicine, University of Genoa
-Biochemistry: School of Dietetic Studies, University of Genova
-Laboratory training for physicians: School of Medicine and Surgery
-Propaedeutic Biochemistry School of Dietetic Studies, University of Genova
-Biochemistry of Cell Signaling: School of Applied and Experimental Biology, University of Genova
-Biochemistry of Nutrition: School of Applied and Experimental Biology, University of Genova
-Supervision of students for experimental master thesis preparation.

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RESEARCH ACTIVITY:

The main subject of investigation concerns the study of the ubiquitous Ca^{2+}-dependent proteolytic system. It includes a family of Ca^{2+}-dependent proteases (calpains) and their natural inhibitor calpastatin (CAST) both localized in cytosol. Calpains do not degrade polypeptides to free amino acids or even to large peptides, but rather act trough limited proteolysis of specific targets. As a consequence the cleaved protein normally retains functional but modified properties. Calpains are involved in many cell functions including signal transduction, cell motility, proliferation, differentiation, and vesicular trafficking. Abnormal activity of calpain has been associated with several pathological conditions characterized by altered Ca^{2+}-homeostasis. In these conditions calpain "shifts" from a physiological to a pathological function and, instead of a limited digestion, the protease produces the complete degradation its targets.

The research focuses on the biochemical mechanisms acting during the activation of the protease. A first aspect concerns the interaction between CAST and calpain. CAST contains four repetitive units that retain inhibitory capacity following calpain-mediated digestion thus providing the first protection against excessive calpain activity. CAST is considered a suicide inhibitor because, after being cleaved by calpain itself to power inhibition of the protease, if Ca^{2+} elevation persists, it is completely degraded. In these conditions calpain is no more regulated, exerts its pathological function and cell damages take place. The final goal of these studies is to limit the abnormal activity of calpain by overexpressing CAST isoforms having a given sequence (especially in the XL-L-DOM called regulatory domain, lacking inhibitory ability) specifically tailored to the cell needs.

A second aspect relates to the functional association of the regulatory subunit of calpain (CSS1) with the corresponding catalytic subunit. Classical calpains are heterodimers consisting of a catalytic subunit unique for each calpain isoform and a common CSS1. The function of CSS1 is to maintain the enzyme in the native condition, and following calpain activation the regulatory subunit is cleaved by calpain at the N-terminus. Recent papers report that overexpression of CSS1 correlates with malignancy of several tumors including glioblastomas. Since native CSS1 seems to stabilize calpain, to explore CSS1 processing could be useful to seek another mechanism for regulating abnormal calpain activity.