UNIVERSITÀ DEGLI STUDI DI PADOVA

DIPARTIMENTO DI SCIENZE BIOMEDICHE -DSB
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Antonio Toninello

Curriculum Vitae

Graduated in Biological Sciences (110/110 *cum laude*), University of Padova, Italy.
1970-1992 Laboratory Assistant; Scientific Collaborator and Researcher of Italian Research Council (CNR), at Unit for Study of Mitochondrial Physiology, Institute of Biological Chemistry, University of Padova.
1992-present Associate Professor of Biological Chemistry, School of Mathematical, Physical and Natural Sciences, Departments of Biochemistry (until 2010) and Department of Biomedical Sciences, University of Padova.
2000-2006 Italian Member at European Commission for COST Actions 917 (Biogenically Active Amines in Food) and 992 (Health implications of Dietary Amines)
2007-2013 Chief of the Biogenic Amines Group of the Italian Biochemical and Molecular Biology Society (SIBBM).
2013-present Senior Scientist named by the Dean of University of Padova
2017-present Member of the Governor Board of the International Polyamines Foundation.

Research Interests and Achievements

The past scientific contributions of Prof. Toninello regard studies on mitochondrial physiology and energy transduction, particularly calcium cycling, and fluxes of magnesium, potassium and phosphate. The results of these studies contributed towards finding the mitochondrial permeability transition (MPT). Prof. Toninello subsequently addressed his interests towards interactions between naturally occurring polyamines and mammalian mitochondria. The most important results of these studies were the discovery in mitochondria of the electrophoretic transport mechanism of polyamines and their prevention of the MPT phenomenon. The interest aroused by these studies led to Prof. Toninello collaborating with prominent national and international laboratories in the field of polyamines. In parallel with these studies, he also carried out investigations on MPT induction by natural compounds and other synthetic antiproliferative agents, the so-called mitochondriotropic agents, identifying mitochondrial targets potentially involved in the intrinsic apoptosis induced by the above compounds. Research in progress in Prof. Toninello's laboratory concerns the phenomenon of signal trasduction to the MPT. The most significant achievements are the discovery, in brain mitochondria, of Tyr-Kinases, belonging to the Src family, and Tyr-phosphorylated proteins such as creatine kinase, hexokinase and adenine nucleotide translocase, all constituents of the structure of the transition pore.

Studies in the above fields resulted in invitations to speak at several meetings in the last decades (Gordon Conferences, European Commission COST Actions (Liege 2000, Krems 2001, Barcelona 2003, Vilnius 2005, Bergen 2006) and, more recently, several International Congresses on Polyamines: S. Petersburg, Heraklion 2013; Istanbul 2014; S. Paulo 2015; Philadelphia 2016; Rome 2017; Taipei 2018; Bangkok 2019.

Organisation:

Prof. Toninello organised the following: International Congress "The Physiological Role of Polyamines", Padova (Italy) 1992; Biogenic Amines: Biochemical, Physiological and Clinical Aspects, Trento (Italy) 2008; Biogenic Amines: Biochemical and Clinical Aspects, Bertinoro (Italy) 2009, Biogenic Amines: Biochemical, Physiological and Clinical Perspectives, Trento (Italy) 2011.
He has served on the Scientific Committees of several International Meetings of Polyamines and organised the annual meetings of the Polyamine Group of the Italian Biochemical Society (2008-present).

International collaborations:

1986-1994: K.D. Garlid. Oregon Graduate Institute of Science and Technology, Portland, Oregon. (Mechanism of polyamine transport in mitochondria)
1998-2001: S. Yagisawa. School of Pharmaceutical Sciences, University of Nagasaki, Japan.
(Energy of polyamine transport)
2000-2003: C. Richter. Swiss Federal Institute of Technology, Zurich, Switzerland.
(Mitochondrial calcium fluxes and redox level)
2005-2008: M. Unzeta. Institut de Neurosciences and Departement de Bioquimica i Biologia Molecular, Universitat Autonoma de Barcelona, Spain.
(Effects of monoamine oxidase inhibitors at mitochondrial and cellular level)
2007-present: A. Khomutov. Engelhardt Institute of Molecular Biology, Russian Academy of Sciences, Moscow.
(Role of agmatine analogues on mitochondrial function)
2004-present: M.P. Marques. Unidade de Quimica-Fisica Molecular, Universidade de Coimbra, Portugal.
(Structural analyses of amine agmatine and correlations with mitochondrial bioenergetics)
2007-present: J. Satriano. Division of Nephrology, UC San Diego and VASDHS, San Diego, California.
(Pathophysiology of agmatine at renal level)
2011-present: Dr. D. E. Edmondson, Department of Biochemistry, Emory University School of Medicine, Atlanta, GA.
(Effect of hydrogen peroxide derived from monoamine oxidase catalysis on mitochondrial function)
2015-present S. Merali (Effects of spermine oxidation in human colon adenocarcinoma cells. Mass spectrometry analyses) School of Pharmacy, Temple University, Philadelphia.
2015-present A Kaiser (Studies on myocytes under hypoxemia in children with complicated falciparum malaria. Apoptotic control by eIF-5A. Medical Research Centre, University of Duisburg-Essen.

Current Research Interests:

Signal transduction to the permeability transition pore
Mitochondriotropic agents in therapeutic treatment against cancer and neurodegeneration
Pathophysiological role of polyamines in mitochondria
Polyamines and Mitochondria Interactions: New therapeutic strategies against cancer.

Key personnel (July 2010)
Antonio Toninello (Group Leader) –Associate professor
Valentina Battaglia –Post-Doc Fellow
Silvia Grancara –PhD students of the PhD program in Biochemistry and Biophysics
Pamela Martinis – PhD students of the PhD program in Biochemistry and Biophysics

Research Topics

Signal transduction to the transition pore
The mitochondrion is now recognized as a multifunctional organelle involved in cellular proliferation, cell death and aging. Deregulation of mitochondrial functions is associated with several pathologies, including neurodegeneration and cancer. This involvement of the mitochondria requires the existence of an integrated system of signaling, to coordinate the various molecular messages which enter and leave the mitochondrion according to the diverse needs of the cell. Protein phosphorylation/dephosphorylation is one of the major systems of signal transduction contribuing to the regulation of the above network. The induction of mitochondrial permeability transition (MPT) plays a fundamental role in mediating the above processes, and Tyr-phoshorylation in particular may contribute to the triggering and regulation of the phenomenon. Recent evidence obtained by our group, in collaboration with Prof. Brunati, has demonstrated the presence in brain mitochondria of Tyr-kinase, belonging to the Src family, in particular Fyn, Src, Lyn, Fgr and Csk (a regulator kinase of this family) and also Tyr-phosphatase Shp-2. In addition, again in our laboratory, the presence of Tyr-phosphorylated proteins such as creatine kinase, hexokinase and adenine nucleotide translocase, all constituents or regulators of the transition pore, has been identified. As other proteins such as glycogen syntase kinase-3β and VDAC, other pore regulators, are Tyr-phosphorylated, identification of the signaling pathway for the induction and regulation of the MPT, mediated by Tyr-phosphorylation, represents an exciting goal for our group.

Mitochondriotropic agents in therapeutic treatment against cancer and neurodegeneration

Interest in mitochondrial diseases and the demonstration that mitochondria play a central role in many non-genetic pathologies have led to a new research approach, aiming at identifying pharmacological tools acting at mitochondrial level.
The strategy consists of the use of drugs able to block (in neurodegenerative diseases) or to activate (in cancer) the process of cell death. The approaches and contexts of this work are manifold, as are the target pathologies. They are based on the discovery of natural

compounds and on preparation of their synthetic derivatives, to favor their transport in mitochondria and improve their functions. These compounds may act as MPT inducers (pro-oxidants) and, consequently, as potential pro-apoptotic and anti-tumoral agents. Alternatively, they may act as MPT inhibitors (anti-oxidants), as potential anti-neurodegenerative drugs or "mild uncouplers" against obesity.
In "mitochondrial medicine", compounds which can specifically address mitochondrial targets, the so-called "mitochondriotropic agents", include polyphenols and their derivatives. Depending on the biological environment, these compounds act as pro-oxidants or anti-oxidants. In particular, at mitochondrial level they may interact with the Fe^{3+} and Cu^{2+} of the respiratory chain and generate reactive oxygen species. Several mitochondriotropic agents are being studied in our laboratory and some of them exhibit significant mitochondrial-mediated anti-proliferative activity. Further studies are in progress in order to synthesize more effective derivates for this activity.

Pathophysiological role of polyamines in mitochondria
Naturally occurring polyamines (spermine, spermidine, putrescine) are physiological polycationic compounds universally known to be essential for biological processes such as cell growth, differentiation, protein synthesis, regulation of ion channel gating, and modulation of oxidative processes. Polyamines are transported in mitochondria by an electrophoretic mechanism exhibiting a non-ohmic force-flux relationship. They can in fact antagonize the action of agents inducing MPT in isolated mitochondria, spermine being one of the most powerful physiological inhibitors. This event has been demonstrated both in cultured cells and in vivo. The exact mechanism of the effect of polyamines is not yet completely understood. Their metabolic pathway has been identified as a target for identifying new antiproliferative drugs, in view of the relationship between intracellular polyamine content and neoplastic cell growth. Studies are in progress in our laboratory to identify all the factors which mediate the action of polyamines at MPT level and, consequently, on apoptosis. Of particular importance are oxidative processes, as polyamines may behave as ROS scavengers. However, due to the activity of polyamine oxidases and spermine oxidase, polyamines may also become producers of ROS. Most probably these opposite effects depend on polyamine concentration and/or the induction of the above enzymes.
A further important investigation regards the phosphorylation of key components of the transition pore. As mentioned above, some tyrosine kinase and Tyr-phosphorylate proteins have been identified in mitochondria, since polyamines are known to activate some kinases (tyrosine kinases and ERK ½). These observations indicate that polyamines plays a prominent role in the regulation of the signal transduction pathway to the transition pore. Very recently it has been demonstrated that the polyamine spermidine can trigger autophagy in yeast, nematodes and flies, extending their lifespan. This is due to inhibition by the polyamine of the enzyme histone acetylase, which regulates the above phenomenon.
The mechanism by means of which spermidine acts in this way is also being studied by our group. We are working to ascertain whether the enzyme spermidine-spermine acetyltransferase (SSAT) can subtract acetylCoA from the activity of histone acetylase, to form acetylspermidine. In addition, as mitophagy and autophagy are correlated with redox stress and MPT induction, and that polyamines may exert significantly different effects on these events, we are interested in ascertaining whether these molecules play other roles in the so-called autophagic switch.

Present collaborations:

Profs. A.M. Brunati and F. Vianello, Dept. of Biological Chemistry, University of Padova, Italy.
Dr. M. Zoratti, CNR Institute of Neuroscience, Padova, Italy.
Prof. E. Agostinelli, Department of Biochemical Sciences, University of Rome “La Sapienza”, Italy.
Profs. S. Colombatto and M.A. Grillo, Department of Medicine and Experimental Biology, University of Turin, Italy.
Dr. J. Satriano, Department of Medicine, University of California, San Diego, U.S.A.
Dr. M.P. Marques, Unit of Molecular Physical Chemistry, University of Coimbra, Portugal.
Dr. A. Khomutov, Engelhardt Institute of Molecular Biology, Russian Academy of Sciences, Moscow.
Dr. D. E. Edmondson, Department of Biochemistry, Emory University School of Medicine, Atlanta, GA .
Dr. Salim Merali and Dr. Carlos Barrero, School of Pharmacy, Temple University, Philadelphia, U.S.A.

Publications 1970-2018

1) TONINELLO A., ZOCCARATO F. (1970) Action of precursors of purine and pyrimidine nucleotides on hepatic lesions caused by ethionine. BOLL SOC ITAL BIOL SPER., vol. 46, p. 715-717.

2) SILIPRANDI D., DE MEIO R.H., TONINELLO A., ZOCCARATO F. (1971) The action of tellurite, a reagent for thiol groups, on mitochondria oxidative processes. BIOCHEM BIOPHYS RES COMMUN., vol. 45, p. 1071-1075.

3) TONINELLO A., ALMIRANTE L. (1973) Incorporation of the precursors of purine and pyrimidine nucleotides in the nucleic acids of rat liver. FARMACO SCI., vol. 28, p. 888-895.

4) SILIPRANDI D., TONINELLO A., ZOCCARATO F., BINDOLI A. (1975) Phosphate transport across the mitochondrial membrane: the influence of thiol oxidation and of Mg++ on inhibition by mercurials. FEBS Lett., vol. 51, p. 15-17.

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7) SILIPRANDI D., TONINELLO A., ZOCCARATO F., RUGOLO M., SILIPRANDI N. (1978) Efflux of magnesium and potassium ions from liver mitochondria induced by inorganic phosphate and by diamide. J BIOENERG BIOMEMBR. vol. 10, p. 1-11.

8) SILIPRANDI D., RUGOLO M., ZOCCARATO F., TONINELLO A., SILIPRANDI N. (1979) Involvement of endogenous phospholipase A2 in Ca2+ and Mg2+ movement induced by inorganic phosphate and diamide in rat liver mitochondria. BIOCHEM BIOPHYS RES COMMUN., vol. 28;88, p. 388-394.

9) SILIPRANDI D., TONINELLO A., GOBBATO S. (1979) Rotenone and oligomycin-like action of trimebutine on liver mitochondria. BIOCHEM PHARMACOL., vol. 28, p. 2675-2677.

10) RUGOLO M., SILIPRANDI D., SILIPRANDI N., TONINELLO A. (1981) Parallel efflux of Ca2+ and Pi in energized rat liver mitochondria. BIOCHEM J., vol. 200, p. 481-486.

11) TONINELLO A., SILIPRANDI D., SILIPRANDI N. (1982) Mg2+ restores membrane potential in rat liver mitochondria deenergized by Ca2+ and phosphate movements. FEBS Lett., vol. 142, p. 63-66.

12) TONINELLO A., SILIPRANDI N. (1982) Restoration of membrane potential in mitochondria deenergized with carbonyl cyanide p-trifluoromethoxyphenylhydrazone (FCCP). BIOCHIM BIOPHYS ACTA, vol. 682, p. 289-292.

13) SILIPRANDI D., SILIPRANDI N., TONINELLO A. (1983) On the relationship between calcium and phosphate transport, transmembrane potential and acetoacetate-induced oxidation of pyridine nucleotides in rat-liver mitochondria. EUR J BIOCHEM., vol. 130, p. 173-175.

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15) TONINELLO A., DI LISA F., SILIPRANDI D., SILIPRANDI N. (1983) On the mechanism of citrate and isocitrate protective action on rat liver mitochondria. BIOCHEM BIOPHYS RES COMMUN., vol. 115, p. 749-755.

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18) TONINELLO A., DI LISA F., SILIPRANDI D., SILIPRANDi N. (1985) Uptake of spermine by rat liver mitochondria and its influence on the transport of phosphate. BIOCHIM BIOPHYS ACTA., vol. 815, p. 399-404.

19) TONINELLO A., DI LISA F., SILIPRANDI D., SILIPRANDI N. (1986) Action of spermine on phosphate transport in liver mitochondria. ARCH BIOCHEM BIOPHYS., vol. 245, p. 363-368.

20) BRANCA D., DI LISA F., SCUTARI G., TONINELLO A., SILIPRANDI N. (1986) Stabilizing action of L-carnitine on the energy-linked processes of mitochondria isolated from perfused rat liver. BIOCHEM PHARMACOL., vol. 35, p. 2839-2841.

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24) TONINELLO A., SILIPRANDI D., CASTAGNINI P., NOVELLO M.C., SILIPRANDI N. (1988) Protective action of methylglyoxal bis (guanylhydrazone) on the mitochondrial membrane. BIOCHEM PHARMACOL, vol. 37, p. 3395-3399.

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29) SILIPRANDI D., BIBAN C., TESTA S., TONINELLO A., SILIPRANDI N. (1992) Effects of palmitoyl CoA and palmitoyl carnitine on the membrane potential and Mg2+ content of rat heart mitochondria. MOL CELL BIOCHEM, vol. 116, p. 117-123.

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