BIOGRAPHICAL SKETCH

NAME: **Pizzo, Paola**
Married, two children

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POSITION TITLE

Ph.D.

Professor of
General Pathology,
University of Padua, Italy

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
University of Padua, Padua, Italy	Bc.S. in Biological Sciences	11/1988	Zoology
University of Padua, Padua, Italy	Ph.D.	12/1993	Experimental Pathology
Weizmann Institute of Science, Rehovot, Israel	Post-doc	12/1994	Neuromuscular Disease

A. Personal Statement

The main scientific interest of Prof. Pizzo regards the mechanisms of Ca^{2+} homeostasis in mammalian cells and the role of Ca^{2+} as second messenger in cellular physiopathology. In the last 15 years she has started a project on Ca^{2+} dysregulation in familial Alzheimer's Disease (FAD). In particular, using different disease cell models, it has been defined how FAD-linked mutated forms of presenilin 2 (PS2) alter cellular Ca^{2+} handling, reducing both intracellular Ca^{2+} store content and mitochondrial Ca^{2+} uptake. Her research pointed out that FAD-PS2, indeed, causes a mitochondrial bioenergetic defect which impacts neuronal functionality. Moreover, her group showed that mutated PS2 is able to increase the tethering between mitochondria and the endoplasmic reticulum (ER), altering different cell processes associated with the domains of tight interaction between the two organelles. Her studies are now aimed to understand whether and how modifications in ER-mitochondria coupling, as well as in the interaction between other organelles within the cell, might be involved in the pathogenesis of the disease. Prof. Pizzo is also involved in research projects dealing with Ca^{2+} handling by different intracellular organelles, using a new family of probes, genetically encoded and based on FRET (Förster Resonance Energy Transfer). She is also involved in the creation and characterization of a new class of dynamic probes for organelle contacts based on the sliptFAST system. She has also several collaborations with different international groups to study the role of Ca^{2+} in diverse physio-pathological processes.

Dr. Pizzo has published more than 100 papers in international journals with an H-index of 54 and total citations of 10164 (by Scopus).

Complete List of Published Work in PubMed: https://www.ncbi.nlm.nih.gov/pubmed/?term=Pizzo+Paola

Google Scholar reference: https://scholar.google.it/citations?user=066JUAgAAAAJ&hl=it

B. Positions and Honors:

1992: Short-term EMBO fellowship recipient at the Weizmann Institute of Science, Rehovot, Israel, working on Duchenne muscular dystrophy.
1993-1994: Telethon fellowship recipient for the study of neuromuscular disease, at the Weizmann Institute of Science, Rehovot, Israel, in the laboratory of Prof. David Yaffe, working on Duchenne muscular dystrophy.
1994-2000: Research assistant at the Department of Biomedical Sciences, University of Padua, working on calcium signaling.

2000-2014: Assistant Professor at the Faculty of Medicine, University of Padua, working on Familial Alzheimer's Disease (FAD) and calcium homeostasis.
2014-2023: Associate Professor in Pathology, School of Medicine, University of Padua.
2014: Positive National Evaluation for Full Professor in Pathology.
2016: Research associate, Neuroscience Institute, CNR, Padua, Italy
2017: Positive National Evaluation for Full Professor in Applied Biology.
2021: Research associate, Study Centre for Neurodegeneration (CESNE), University of Padua.
2023-present: Full Professor in General Pathology, School of Medicine, University of Padua.

C. Selected peer-reviewed publications

-Giacomello M., Barbiero L., Zatti G., Squitti R., Binetti G., Pozzan T., Fasolato C., Ghidoni R. and **Pizzo P.** (2005). "Reduction of Ca^{2+} stores and Capacitative Ca^{2+} Entry is associated with the Familial Alzheimer's Disease presenilin-2 T122R mutation and anticipates the onset of dementia". *Neurobiology of Disease*, 18: 638-648.
-Zatti G., Burgo A., Giacomello M., Barbiero L., Guidoni R., Sinigaglia G., Florean C., Bagnoli S., Binetti G., Sorbi S., **Pizzo P.*** and Fasolato C.* (*corresponding author) (2006) "Presenilin mutations linked to Familial Alzheimer's Disease reduce endoplasmic reticulum and Golgi apparatus calcium levels". *Cell Calcium*, 39:539-550.
-Brunello L., Zampese E., Florean C., Pozzan T., **Pizzo P.*** and Fasolato C.* (*corresponding author) (2009). "Presenilin-2 dampens intracellular Ca^{2+} stores by increasing Ca^{2+} leakage and reducing Ca^{2+} uptake". *J Cell Mol Med.*, 13:3358-69.
-Giacomello M., Drago I., Bortolozzi M., Scorzeto M., Gianelle A., **Pizzo P.** and Pozzan T. (2010). "Ca2+ hot spots on the mitochondrial surface are generated by Ca2+ mobilization from stores, but not activation of store operated Ca2+ channels". *Molecular Cell*, 38:280-90.
-Zampese E., Fasolato C., Kipanyula M.J., Bortolozzi M., Pozzan T. and **Pizzo P.** (2011). "Presenilin 2 modulates ER-mitochondria interactions and Ca^{2+} cross-talk". *Proc Natl Acad Sci U S A.*, 108: 2777-82.
-Hedskog L., Pinho C.M., Filadi R., Rönnbäck A., Hertwig L., Wiehager B., Larssen P., Gellhaar S., Sandebring A., Westerlund M., Graff C., Winblad B., Galter D., Behbahani H., **Pizzo P.**, Glaser E., Ankarcrona M. (2013) Modulation of the endoplasmic reticulum-mitochondria interface in Alzheimer's disease and related models. *Proc Natl Acad Sci U S A*, 110(19):7916-21
-Filadi R., Greotti E., Turacchio G., Luini, A., Pozzan T., **Pizzo P.** (2015) Mitofusin 2 ablation increases endoplasmic reticulum-mitochondria coupling. *Proc Natl Acad Sci USA* 112(17):E2174-81. Evaluated and recommended by FACULTY OF 1000.
-Filadi R., Greotti E., Turacchio G., Luini, A., Pozzan T., **Pizzo P.** (2016) Presenilin 2 modulates endoplasmic reticulum-mitochondria coupling by tuning the antagonistic effect of mitofusin 2. *Cell Reports*,15(10):2226-38.
-Filadi R., Leal N.S., Schreiner B., Rossi A., Dentoni G., Moreira Pinho C., Wiehager B., Cieri D., Calì T., **Pizzo P.*** and Ankarcrona M.* (*corresponding author) (2018) TOM70 sustains cell bioenergetics by promoting IP3R3-mediated ER to mitochondria Ca2+ transfer. *Current Biology*, 28(3): 369-382.e6.
-Theurey P., Connolly N.M., Fortunati I., Basso E., Lauwen S., Ferrante C., Moreira Pinho C., Joselin A., Gioran A., Bano D., Park D., Ankarcrona M., **Pizzo P**.* and Prehn J.H.M.* (*corresponding author) (2019) Systems biology analysis identifies impairment of mitochondrial and glycolytic metabolism in a genetic model of Alzheimer's disease. *Aging Cell*, 18(3):e12924.
-Fedeli C., Filadi R., Rossi A., Mammucari C. and **Pizzo P.** (2019) PSEN2 (presenilin 2) mutants linked to familial Alzheimer disease impair autophagy by altering Ca^{2+} homeostasis. *Autophagy*, 20:1-19.
-Rossi A., Rigotto G., Valente G., Giorgio V., Basso E., Filadi R. and **Pizzo P.** (2020). Defective mitochondrial pyruvate flux affects cell bioenergetics in Alzheimer's disease related models. *Cell Reports*, 30(7):2332-2348.e10.
-Filadi R, De Mario A, Audano M, Romani P, Pedretti S, Cardenas C, Dupont S, Mammucari C, Mitro N, **Pizzo P** (2023). Sustained IP3-linked Ca2+ signaling promotes progression of breast cancer cells by regulating fatty acid metabolism. *Frontiers in Cell and Developmental Biology*, doi: 10.3389/fcell.2023.1071037.
- Garcia Casas P, Rossini M, Påvénius L, Saeed M, Arnst N, Sonda S, Bruzzone M, Berno V, Raimondi A, Sassano ML, Naia L, Agostinis P, Sturlese M, Niemeyer B, Brisman H, Ankarcrona M, Gautier A, **Pizzo P***, Filadi R* (*corresponding autor). (2024) Simultaneous detection of membrane contact dynamics and associated Ca2+ signals by reversible chemogenetic reporters. *Nat Comm, 15(1):9775.*

D. Ongoing Research Support:

1. Cure Alzheimer's Fund (USA), 2022-2025, "Extracellular ATP is a key factor in promoting Alzheimer's disease neuroinflammation". The project aims at defying the contribution of the extracellularATP-P2X7 receptor axis in sustaining neuroinflammation in AD. co-PI, 645.000 USD.
2. Next Generation EU and Italian Ministry of Research, 2022-2024, NRRP-National Recovery and Resilience Plan grant, National Centre of Research "Development of gene therapy and drugs with RNA technology", spoke 3 "Neurodegenerative Diseases", task 3.2.6 "Targeting neuroinflammation". PI, 400.000 euro.
3. Alzheimer's Association (USA), 2024-2026, "Organelle contact sites as determinants of endolysosomal activity in AD". The project aims at defying alteration of organelle contact sites in endolysosomal activity of AD experimental models. PI, 298.500 USD
4. Italian Ministry of Research Grant (PRIN2022), 2022-2023, "Purinergic checkpoints in neuroinflammation and Alzheimer's disease: extracellular ATP and the P2X7 receptor as main drivers of neurodegeneration". The project aims at defying the contribution of the extracellular ATP-P2X7 receptor signaling axis in sustaining neuroinflammation in AD. PI, 336.000 euro.
5. Italian Ministry of Research Grant (PRIN_PNRR2022), 2022-2023, "Deciphering membrane contacts in neurodegenerative disorders by dynamic chemogenetic reporters". The project aims at studying organelle contact sites in different neurodegenerative diseases by a new family of dynamic fluorescent probes based on the split-FAST system. Co-PI, 279.499 euro.