Valeria Magnelli

DATI ANAGRAFICI

Born in Genova on February, 21st 1964. Living in Genova

CURRICULUM VITAE ET STUDIORUM

- Scientific high school degree – 1984
- Degree in Scienze Naturali, University of Genova, on December 5th, 1988, *summa cum laude*. Thesis discussion "Carachetrization and purification of retinal isomerase from Apis mellifera exctract"
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- Degree in Scienze Biologiche, University of Genova, on March 8th, 1990, *summa cum laude*. Thesis discussion: "Anaesthetic effect on chloride currents of $GABA_A$ complex, in primary cultured cells"

ACADEMIC CAREER

1999 -	University Researcher ssd BIO/09 (Fisiologia Generale) at Dipartimento di Scienze e Innovazione Tecnologica University of Piemonte Orientale, Alessandria
1995	Visiting researcher at Max Planck Institut für Psychiatrie di Monaco
1993 - 1999	University reseracher ssd BIO/09 (Fisiologia Generale) at Facoltà di Farmacia University di Torino, in Novara
1992 - 1993	PhD in Neurobiology and Neurophysiology at University of Ferrara
1991 - 1992	Fellow at Dept. Of Molecular Physiology and Biophysics at Texas Medical Center, Houston (Texas, USA)
1989 - -1991	Fellow at "Centro di Ricerca Neuromuscolare" in Dept. of Anesthesia and Intensive Care of Ospedale Civile in Sondrio

RESEARCH FIELDS

1. Calcium channels and Fura evaluation of intracellular calcium levels
2. Prostate cells and androgen effect in methastasis progression
3. Electrophysiology and patch-clamp
4. Antioxidant molecules

RESEARCH ACTIVITY

1. **Role of calcium in phenotype modulation in prostate cells resistant to androgen treatment**

Androgen receptor plays a crucial role in growth and proliferation of normal prostatic cells. It has been involved in the development of the main forms of prostate cancer.
Androgen deprivation is the main focus treatment in prostate cancer therapy and it has been seen that this often causes a consistent regression of the cancer. Despite of this, in some patients the cancer goes towards very aggressive phenotypes which haven't valid therapies. The transition between the phenotypes may be driven by membrane calcium channels, whose role is still poorly investigated. My research wants to focus on the possible mechanisms involved in this transition and evaluate if calcium dynamics can antagonize this process. We will evaluate the different expression of calcium-dependent protein and calcium channels in different cell lines with increasing malignity.

ACADEMIC COMMISSIONS

2015 –	Referent for Erasmus at DISIT
2010 – 2015	Referent for International Commission at DISIT

LIST OF SIGNIFICANT PUBLICATIONS

- Resveratrol induces intracellular Ca(2+) rise via T-type Ca(2+) channels in a mesothelioma cell line. *Marchetti C, Ribulla S,* ***Magnelli V****, Patrone M, Burlando B.* Life Sci. 2016 148:125-31
- Epigallocatechin-3-gallate elicits Ca2+ spike in MCF-7 breast cancer cells: essential role of Cav3.2 channels. *Ranzato E,* ***Magnelli V****, Martinotti S, Waheed Z, Cain SM, Snutch TP, Marchetti C, Burlando B.*
- Cell Calcium. 2014 56:285-95.
- Epigallocatechin-3-gallate induces mesothelioma cell death via H2 O2 -dependent T-type Ca2+ channel opening. *Ranzato E, Martinotti S,* ***Magnelli V****, Murer B, Biffo S, Mutti L, Burlando B.* J Cell Mol Med. 2012 16:2667-78

- Antagonists-resistant calcium currents in rat embryo motoneurons. ***Magnelli V****, Baldelli P, Carbone E.* Eur J Neurosci. 1998 10:1810-25

- The action of amyotrophic lateral sclerosis immunoglobulins on mammalian single skeletal muscle Ca2+ channels. ***Magnelli V,*** *Sawada T, Delbono O, Smith RG, Appel SH, Stefani E*. J Physiol. 1993 461:103-18