europass

# Martina Vidmar

## PRESENTAZIONE

Dottoranda in Scienze della Riproduzione e dello Sviluppo

## ISTRUZIONE E FORMAZIONE

01/11/2020 – ATTUALE Trieste, Italia

**DOTTORATO IN SCIENZE DELLA RIPRODUZIONE E DELLO SVILUPPO** Università degli Studi di Trieste

Il dottorato mi ha permesso di imparare a gestire in modo autonomo un progetto di ricerca e di apprendere o perfezionare tecniche di laboratorio quali: lavoro con le cellule, trasfusione cellulare, disegno di plasmidi e clonaggi, staining di immunofluorescenza, proximity ligation assay, western blot, estrazione di acidi nucleici, qPCR.

01/10/2016 – 22/03/2019 Trieste, Italia

**LAUREA MAGISTRALE IN BIOTECNOLOGIE MEDICHE E FARMACEUTICHE** Università degli Studi di Trieste

Il tirocinio finalizzato per la tesi magistrale l'ho effettuato nel laboratorio del Dr. Andy Baker, Edimburgo (UK), esperienza che mi ha permesso di imparare diverse tecniche di biologia molecolare, nonché migliorare il mio livello di inglese.

**Voto finale** 110/110 | **Tesi** Role of miRNA-183 cluster in cardiac regeneration

01/10/2013 – 21/12/2018 Trieste, Italia

**LAUREA TRIENNALE IN SCIENZE E TECNICHE BIOLOGICHE** Università degli Studi di Trieste

**Voto finale** 104/110

## ESPERIENZA LAVORATIVA

Trieste, Italia

**COLLABORATORE FAMILIARE/COLLABORATRICE FAMILIARE** CODAQ

-sviluppo e gestione del sito web
-gestione database

## COMPETENZE DIGITALI

Buon utilizzo di MS – Office: Word, Excel, Power Point, Outlook etc. | Librerie Python(pandas, numpy, scipy, Scikit-learn, Spacy, Matplotlib,Tensorflow..) | Creazione di siti web con CMS (Wordpress, Joomla,Prestashop)

## COMPETENZE LINGUISTICHE

Lingua madre: **ITALIANO**

Altre lingue:

| | COMPRENSIONE | | ESPRESSIONE ORALE | | SCRITTURA |
|---|---|---|---|---|---|
| | Ascolto | Lettura | Produzione orale | Interazione orale | |
| **INGLESE** | C1 | C1 | C1 | C1 | C1 |

*Livelli: A1 e A2: Livello elementare B1 e B2: Livello intermedio C1 e C2: Livello avanzato*

## ULTERIORI INFORMAZIONI

### CONFERENZE E SEMINARI

17/09/2022 – 21/09/2022 – Napoli
**Congresso SIM 2022** Partecipazione al congresso con un poster intitolato: "Role of Oral and Gut Microbiota in pediatric Autism Spectrum Disorder"

### PUBBLICAZIONI

**Mir-96 and miR-183 differentially regulate neonatal and adult post-infarct neovascularisation. // Raphael F.P. Castellan, Milena Vitiello, Martina Vidmar, Steven Johnstone, Dominga Iacobazzi, David Mellis, Benjamin Cathcart, Adrian JW Thomson, Christiana Ruhrberg, Massimo Caputo, David E. Newby, Gillian A. Gray, Andrew Howard Baker, Andrea Caporali, Marco Meloni.**

– 2020
ScFollowing myocardial infarction (MI), the adult heart has minimal regenerative potential. Conversely, the neonatal heart can undergo extensive regeneration, and neovascularization capacity was hypothesized to contribute to this difference. Here, we demonstrate the higher angiogenic potential of neonatal compared with adult mouse cardiac endothelial cells (MCECs) in vitro and use this difference to identify candidate microRNAs (miRs) regulating cardiac angiogenesis after MI. miR expression profiling revealed miR-96 and miR-183 upregulation in adult compared with neonatal MCECs. Their overexpression decreased the angiogenic potential of neonatal MCECs in vitro and prevented scar resolution and neovascularization in neonatal mice after MI. Inversely, their inhibition improved the angiogenic potential of adult MCECs, and miR-96/miR-183–KO mice had increased peri-infarct neovascularization. In silico analyses identified anillin (ANLN) as a direct target of miR-96 and miR-183. In agreement, Anln expression declined following their overexpression and increased after their inhibition in vitro. Moreover, ANLN expression inversely correlated with miR-96 expression and age in cardiac ECs of cardiovascular patients. In vivo, ANLN+ vessels were enriched in the peri-infarct area of miR-96/miR-183–KO mice. These findings identify miR-96 and miR-183 as regulators of neovascularization following MI and miR-regulated genes, such as anillin, as potential therapeutic targets for cardiovascular disease.

**Trichoplein Binds PCM1 and Controls Endothelial Cell Function by Regulating Autophagy. //Andrea Martello , Angela Lauriola , David Mellis , Elisa Parish , John C Dawson, Lisa Imrie, Martina Vidmar , Noor Gammoh , Tijana Mitić , Mairi Brittan , Nicholas L Mills , Neil O Carragher , Domenico D'Arca , Andrea Caporali.**

– 2020
Scrivi qui la dAutophagy is an essential cellular quality control process that has emerged as a critical one for vascular homeostasis. Here, we show that trichoplein (TCHP) links autophagy with endothelial cell (EC) function. TCHP localizes to centriolar satellites, where it binds and stabilizes PCM1. Loss of TCHP leads to delocalization and proteasome-dependent degradation of PCM1, further resulting in degradation of PCM1's binding partner GABARAP. Autophagic flux under basal conditions is impaired in THCP-depleted ECs, and SQSTM1/p62 (p62) accumulates. We further show that TCHP promotes autophagosome maturation and efficient clearance of p62 within lysosomes, without affecting their degradative capacity. Reduced TCHP and high p62 levels are detected in primary ECs from patients with coronary artery disease. This phenotype correlates with impaired EC function and can be ameliorated by NF-κB inhibition. Moreover, Tchp knock-out mice accumulate of p62 in the heart and cardiac vessels correlating with reduced cardiac vascularization. Taken together, our data reveal that TCHP regulates endothelial cell function via an autophagy-mediated mechanism.