TRP expression signature in tumor-derived endothelial cells: functional roles in prostate cancer angiogenesis

Michela Bernardini1,2,3, Giorgia Chinni1,2, Guillaume Grolez1,2,3, Alessia Brossa4, Giulia Trimaglio2,3, Laurent Allart2,3, Audrey Hulot5, Guillemette Marot7,8, Tullio Genova1, Aditi Joshi1, Virginie Mattot5, Gaelle Fromont-Hankradi, Fabrice Soncin6, Benedetta Bussolati4, Natalia Prevorskaya1,2, Luca Munaron5, Alessandra Fiorio Pla1,2,3 & Dimitra Gkika1,2

1Department of Life Science and Systems Biology, University of Torino, Turin, Italy. 2Inserm U1003, Université Lille 1, Villeneuve d’Ascq, France. 3Laboratory of Excellence, Ion Channels Science and Therapeutics, Université de Lille 1, Villeneuve d’Ascq, France. 4Department of Molecular Biotechnology and Health Sciences, Molecular Biotechnology Centre, University of Turin, Turin, Italy. 5Inserm UMR 1069, Université de Tours, Tours, France. 6Institute de Biologie de Lille, Lille, France. 7Univ. Lille, Institut Français de Bioinformatique, b/lle, F-59000 Lille, France. 8Univ. Lille, Inria, CHU Lille, EA 2694-MODAL-Models for Data Analysis and Learning, F-59000 Lille, France.

INTRODUCTION

TRP channels play a key role in cancer progression, modulating cell proliferation and survival, cancer invasion of surrounding tissues and angiogenesis. TRP expression could therefore characterize the prostate cancer (PCa) cell phenotype. Another well-established concept is that TRPs deeply modulate endothelial cell (EC) biology and tumor angiogenesis. However, a specific TRP expression signature of PCa angiogenesis is still lacking.

Our aim was to profile the expression of TRP channels during PCa angiogenesis and then to identify the specific molecular modulators of this process proving novel therapeutic targets.

CONCLUSION

It was previously shown that PTEC exhibit the aggressive phenotype typical of TECs (Fiorio Pla et al. 2014). Here we identified three ‘prostate specific’ TEC overexpressed TRPs: TRPV2, TRPC3 and TRPA1 involved in different aspects of the angiogenic process. Taken together, our expression profiling and functional data could explain the transition of prostate endothelial cells to their aggressive tumor phenotype, proposing novel molecular players to selectively target PCa progression and angiogenesis. Results recently published (Bernardini et al., Cancers 2019).