

This is an Open Access document downloaded from ORCA, Cardiff University's institutional repository: <https://orca.cardiff.ac.uk/id/eprint/100349/>

This is the author's version of a work that was submitted to / accepted for publication.

Citation for final published version:

Moriconi, Chiara , Palmieri, Valentina, Tornillo, Giusy, Fillmore, Helen, Pilkington, Geoff and Gumbleton, Mark 2017. Caveolin-1 implicated as a pro-invasive gene in high-grade glioma cell models: implementation of a 3d spheroid matrix invasion assay. *Neuro-Oncology* 19 (S1) , i21-i22. 10.1093/neuonc/now293.080

Publishers page: <https://doi.org/10.1093/neuonc/now293.080>

Please note:

Changes made as a result of publishing processes such as copy-editing, formatting and page numbers may not be reflected in this version. For the definitive version of this publication, please refer to the published source. You are advised to consult the publisher's version if you wish to cite this paper.

This version is being made available in accordance with publisher policies. See <http://orca.cf.ac.uk/policies.html> for usage policies. Copyright and moral rights for publications made available in ORCA are retained by the copyright holders.



PP80. CAVEOLIN-1 IMPLICATED AS A PRO-INVASIVE GENE IN HIGH-GRADE GLIOMA CELL MODELS: IMPLEMENTATION OF A 3D SPHEROID MATRIX INVASION ASSAY

Ms Chiara Moriconi, Dr Valentina Palmieri, Dr Giusy Tornillo, Dr Helen Fillmore, Prof Geoff Pilkington, Prof Mark Gumbleton;
Cardiff University/ School of Pharmacy and Pharmaceutical Sciences

INTRODUCTION: The poor prognosis associated with Glioblastoma multiforme (GBM) is multifactorial but includes the capacity of residual tumour cells not removed by surgery and resistant to radio-/chemo-therapy undergoing diffuse invasion into the surrounding brain tissue. Caveolin-1 (Cav-1) is the major structural and functional component of caveolae. In a number of tumour types Cav-1 is recognised to participate in cytoskeletal rearrangement, integrin-mediated adhesion and/or matrix remodelling. We proposed Cav-1 serves to promote invasion of GBM cells. To investigate this we have employed in an in-vitro 3D cellular invasion assay.

METHOD: The human GBM cell lines, UP007 and UP029 established from primary cultures of biopsy-derived brain tumours (University of Portsmouth), U-87 MG (ECACC) and U-373 MG (ECACC) were genetically modified to stably knock-out Cav-1 using a Lentiviral Cav-1 shRNA approach; corresponding stably transfected non-target (NT) shRNA cell lines were generated as controls. Neurospheres were formed and embedded within an extracellular matrix (Matrigel™). Over a two-/four-day period (depending on cell line) the migration of cells away from the neurosphere core (CORE) was quantified by image capture and processing (Image J) using a custom-developed MatLab script for pixel density analysis indicative of the density of migrating cellular material.

RESULTS: Cav-1 knockout resulted in significant (P0.05) towards reduced invasion. Depending upon the cell line the Cav-1 knockdown also resulted in reduced size and cellular density of the neurosphere core (UP007 and UP029) indicative of reduced proliferation and/or cell survival capacity. Abstracts i22 NEURO-ONCOLOGY • January 2017

CONCLUSION: Using an in-vitro 3D cellular invasion assay we have found Cav-1 expression in a series of three GBM cell lines to promote cellular invasion capacity. Ongoing studies are addressing signalling mechanisms and the influence of the microenvironment.

ACKNOWLEDGEMENTS: CM and MG acknowledge support from the Cancer Research Wales Ed Evans Scholarship for CM. GP and HF acknowledge support from Brain Tumour Research.