Dr Jessica Edwards BSc (Hons); MSc; PhD

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About

Jessica Edwards is a recent PhD graduate now working in Professor Paul Murrays Group researching Brain Tumours. Her PhD was conducted in Wound Healing Research investigating interactions of the Transglutaminase family and their effects on the extracellular matrix during re-epithelialisation.

Now employed by Birmingham University she hopes her background in signalling and co culture modelling will aid investigation of the tumour microenvironment and its interactions causing pathology of Brain Cancer. Jessica has presented her PhD work at local, national and International level.

Qualifications

- Research Associate, University of Birmingham
- PhD Cell and Matrix Biology, Cardiff University 2010
- MSc Human Genetics, University of Leeds 2002
- BSc (Hons) Genetics, University of Wales, Aberystwyth.

Biography

Jessica Edwards qualified with a BSc (Hons) in Genetics from University of Wales, Aberystwyth in 2000. She then went on to complete an MSc in Human Genetics from Leeds University in 2002. The following years were spent in a mixture of commercial and NHS pathology labs before returning to academia to complete a PhD in Cell and Matrix Biology at Cardiff University finished in 2010. Her PhD was based on Transglutaminase 2 regulating epithelial signalling responses in wound healing.

Currently employed in Professor Paul Murrays group as a Research Associate Jessica is a keen and enthusiastic young researcher. Jessica's main research interests being cell signalling, and understanding the cellular and molecular mechanisms that drive brain cancer pathology and progression - with the ultimate aim of manipulating this understanding to develop therapeutic strategies.

Research

Malignant brain tumours, particularly the intrinsic glioma variety (Glioblastoma), are among the most aggressive and fatal types of cancer in man. For example, most patients with Glioblastoma succumb to the disease within 2 years of diagnosis and there are no long-term survivors. Jessica Edwards under the direction of Professor Paul Murray and Dr Allah Detta investigates samples from both primary and secondary brain tumours.

Separation of the different cell types allows study of their individual properties and collectively in co-culture models to discover target protein or gene signalling pathways relevant to the pathogenesis of brain cancer.

MIF has been identified as a potential novel therapeutic target. The macrophage inhibition factor (MIF) is a pro-inflammatory cytokine first described as a soluble factor produced by T lymphocytes that can activate macrophages while at the same time inhibiting their migration. As well as playing a role in host inflammatory and immune responses, it has been reported to be over-expressed in different tumour types where it has been shown to regulate cell proliferation, survival and angiogenesis and to promote tumorigenesis by inhibiting p53 accumulation. MIF has been shown to be over-expressed in glioblastoma (Markert et al., Physiol Genemics 5:21-33; 2001) and several other studies have implicated MIF in its pathogenesis. For example, Piette et al (J BIol Chem 284:32483-32492; 2009) reported that in glioblastoma cell lines MIF increases migration and invasion through stimulation of the ERK1/2 MAP kinase pathway upon phosphorylation of ERK1/2 following its endocytosis or binding with CD74 ___ its receptor.

Binding of CD74 induces a signalling cascade which increases proliferation, migration and invasion. ERK1/2 MAP kinase has also been shown to be upregulated following epidermal growth factor receptor amplification and mutation (frequent in gliomas), leading to induction of the urokinase type plasminogen activator and its receptor, resulting in increased cell migration in squamous cell carcinoma cells, modulation of matrix metalloproteinase activity in vascular smooth muscle cells, enhancement of α1β1 integrin expression in gastric carcinoma cells, and alteration of the cytoskeleton in neuroblastoma cells. However, since the mechanisms of these pro-proliferative and pro-migratory and pro-invasion signalling cascades are not understood in Glioblastoma, they can all be potentially useful future drug targets or inhibitory targets for MIF or EGFR inhibitors once their role in glioblastoma tumourigenesis has been established.