Postdoctoral Scientist – Dr Angeliki Malliri, Cell Signalling Group

- Salary in the range of £30,000 - £39,800 (dependent upon qualifications and experience)
- Job Ref: MI/15/83
- 3 year position

A 3 year position is available in the laboratory of Dr Angeliki Malliri. The overall aim of her group is to investigate the role of Rho proteins like Rac, found at high levels and mutated in cancer, in cancer cell motility, adhesion and survival. The successful applicant will study the role of the Rac activator Tiam1 and its homologue Tiam2/STEF in the development and progression of lung cancer using recently generated *in vivo* models.

The successful applicant should have a PhD in molecular and cellular biology or related discipline. A background working with *in vivo* models is highly desirable.

The Cancer Research UK Manchester Institute (www.cruk.manchester.ac.uk), an Institute of The University of Manchester (www.manchester.ac.uk), is a world-leading centre for excellence in cancer research. The Institute, core-funded by Cancer Research UK (www.cancerresearchuk.org), the largest independent cancer research organisation in the world, has exceptional laboratory facilities and state-of-the-art core services, including next generation sequencing, flow cytometry, confocal microscopy, bioinformatics, histology and mass-spectrometry. We are adjacent to The Christie NHS Foundation Trust (www.christie.nhs.uk), one of the largest cancer treatment centres in Europe. These factors combine to provide an exceptional environment in which to pursue basic, translational and clinical research programmes in a vibrant and dynamic city surrounded by beautiful countryside and with excellent national and international transport links.

Recent highlight publications from Cell Signalling lab include:

Whalley et al. (2015) Cdk1 phosphorylates the Rac activator Tiam1 to activate centrosomal Pak and promote mitotic spindle formation. *Nat Commun.* 6, 7437.
Vaughan et al. (2015) HUWE1 ubiquitylates and degrades the RAC activator TIAM1 promoting cell-cell adhesion disassembly, migration, and invasion. *Cell Rep*. 6, 88.
Mack et al. (2012) β2-syntrophin and Par-3 promote an apicobasal Rac activity gradient at cell-cell junctions by differentially regulating Tiam1 activity. *Nature Cell Biology* 14, 1169.
Castillo-Lluva et al. (2010) SUMOylation of the GTPase Rac1 is required for optimal cell migration. *Nature Cell Biology* 12, 1078.
Woodcock et al. (2010) Tiam1-Rac signaling counteracts Eg5 during bipolar spindle assembly to facilitate chromosome congression. *Curr Biol*. 20, 669.
Woodcock et al. (2009) Src-induced disassembly of adherens junctions requires localized phosphorylation and degradation of the Rac activator Tiam1. *Molecular Cell* 33, 639.

Informal enquiries should be directed to Dr Angeliki Malliri via email: Angeliki.Malliri@cruk.manchester.ac.uk

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Closing date: 16th December 2015