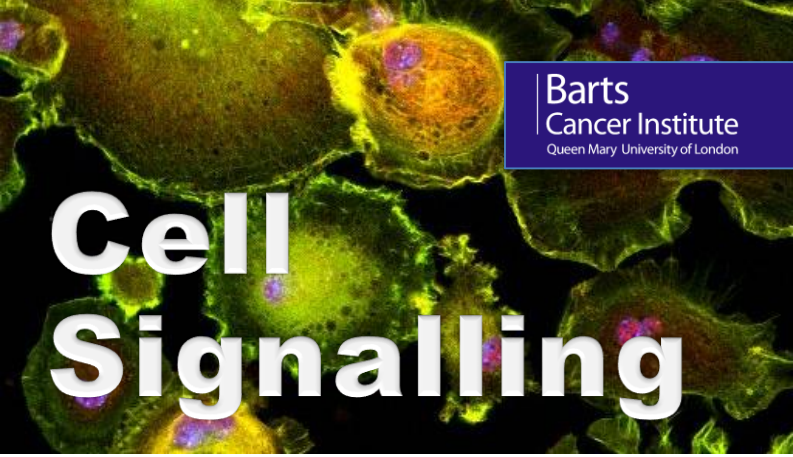
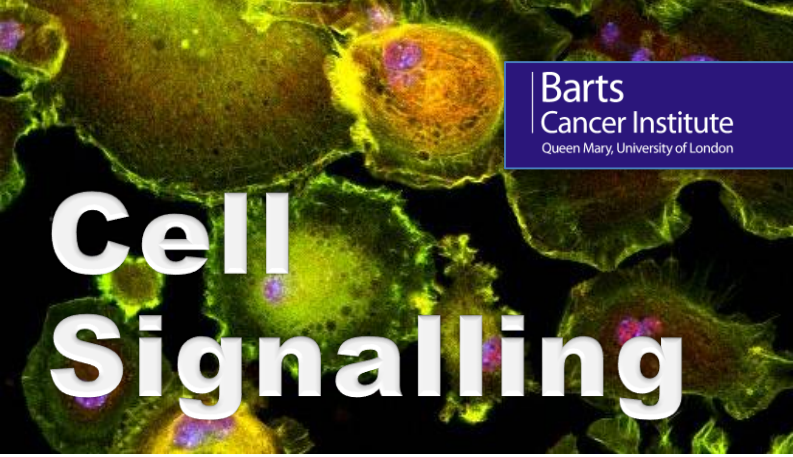



## Why we focus on Cell Signalling

Cell signalling is the process by which our cells communicate with each other and with their environment. Pathways that govern how cells proliferate, move, survive and differentiate represent key targets in cancer biology. Targeting cell signalling has proven extremely successful in cancer treatment – drugs such as *Glivec*, *Herceptin* and *Iressa*, all discovered as a result of our increased understanding of cell signalling, are being used to treat millions of patients worldwide.

## What we do

- We use novel proteomic approaches to study cell signalling pathways in unprecedented detail, allowing us to understand chemoresistance and cancer cell metabolism.
- We study compartmental signalling and how receptors, in particular c-Met, are trafficked within cancer cells, thus identifying novel pathways and therapeutic targets.
- We study Fibroblast Growth Factor Receptors and their impact on cancer cell behaviour, focusing on their nuclear trafficking and their oncogenic mutations.
- We study the role of voltage-gated proton channels in B-cell cancers and their role in supporting B Cell Receptor signalling.
- We study novel aspects of members of the LIMD1 family, which play key tumour supressive roles in epithelial cancers.




## Key Publications


- Receptor Tyrosine Kinase c-Met controls the cytoskeleton from different endosomes via different pathways. Ménard L, Parker PJ, Kermorgant S. (2014) *Nature Communications* , 5:3907
- Coleman *et al*. Pancreatic cancer cell invasion is mediated by nuclear translocation of FGFR1 and FGF2 in stellate cells. *EMBO Mol Med*. 2014; 6:467-481.
- Foxler *et al.* The LIMD1 protein bridges an association between the prolyl hydroxylases and VHL to repress HIF-1 activity. *Nat Cell Biol* 2012; 14(2):201-8.
- Beltran *et al*. Calpain interacts with class IA phosphoinositide 3-kinases regulating their stability and signaling activity. *PNAS USA* 2011; 108: 16217-22.
- Capasso *et al.* HVCN1 modulates BCR signal strength via regulation of BCR-dependent generation of reactive oxygen species. *Nat Immunol* 2010; 11(3):265-72.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