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First person – Elliott Bernard

First Person is a series of interviews with the first authors of a selection of papers published in Journal of Cell Science, helping early-career researchers promote themselves alongside their papers. Elliott Bernard is first author on '*M. tuberculosis* infection of human iPSC-derived macrophages reveals complex membrane dynamics during xenophagy evasion', published in JCS. Elliott is a PhD student in the lab of Maximiliano (Max) Gutierrez at The Francis Crick Institute, London, UK, investigating the cell biology of the endolysosomal and autophagy systems and their dysfunction in disease.

Elliott Bernard

How would you explain the main findings of your paper in lay terms?

Mycobacterium tuberculosis (Mtb), the bacterium that causes tuberculosis (TB), infects lots of different cell types in the body. One of the most important cell types is an immune cell called the macrophage. Macrophages normally hoover up bacteria, viruses and dead cells to keep you healthy. Mtb sets up home in these cells where it then grows and can cause the cell to die. As part of its life, some Mtb damage the membrane of the phagosome – the compartment that houses the bacteria inside the cell – to access the cytosol. Macrophages, and other cells, use a process called autophagy to try to prevent bacteria, including Mtb, accessing the cytosol, which might be a particularly good place for the bacteria to grow. My work, using a human macrophage model derived from stem cells, revealed the dynamics of the autophagy process during Mtb infection and showed that the bacteria are able to escape from the autophagy machinery and successfully get into the cytosol. We also identify how human macrophages respond by altering gene expression when infected with Mtb or an attenuated mutant of the bacteria that are unable to damage the phagosome.

Were there any specific challenges associated with this project? If so, how did you overcome them?

The biggest challenge was making the live-cell movies. As we work with a virulent human pathogen, all the work has to be completed under BSL3 conditions, which brings with it a unique set of challenges. We are lucky enough to have a couple of microscopes in our BSL3 facility that allow live-cell imaging – something only a few TB labs in the world can do. Once the dish is on the microscope, the challenge is selecting the best cells, with a good expression level of the constructs and then hoping they become infected! But in the end the hard work was very worthwhile.

When doing the research, did you have a particular result or 'eureka' moment that has stuck with you?

Absolutely! The first time I got the macrophages to express GFP–LC3B – a reporter for monitoring the autophagy machinery – and infected them with Mtb to image live. It revealed the remarkable, and very unexpected, large autophagic structures generated upon membrane damage by Mtb for the first time. This was a movie Max had been waiting for since discovering a role for autophagy in defending cells from Mtb during his PhD over 15 years ago. We went to the bar and had G&Ts to celebrate!

Why did you choose Journal of Cell Science for your paper?

We heard about the planned special edition on host–pathogen interactions (via Twitter, of course!) and knew our paper could fit really nicely into this. With its great reputation for publishing interesting cell biology for a broad readership, Journal of Cell Science was a great place to publish our paper to tell the community about our discovery. We were also able to use the fast track option with previous peer review, making the submission process super simple and quick.

What motivated you to pursue a career in science, and what have been the most interesting moments on the path that led you to where you are now?

I've always been inquisitive and wanted to learn and understand more about how the world, and especially the human body, works. Science, mostly biology and chemistry, were subjects I enjoyed throughout school and that set me on the path to study Biochemistry for my undergraduate degree. During this, I spent a year in Max's lab as a sandwich student working on a Rab protein in the autophagy pathway and then a few months in my final year in Jon Lane's lab at the University of Bristol. Both of these experiences showed me how much fun there was to be had in the lab, discovering and seeing things for the first time. That's what still gets me excited every day I am in the lab – the thought that I might find something that no one has seen before.

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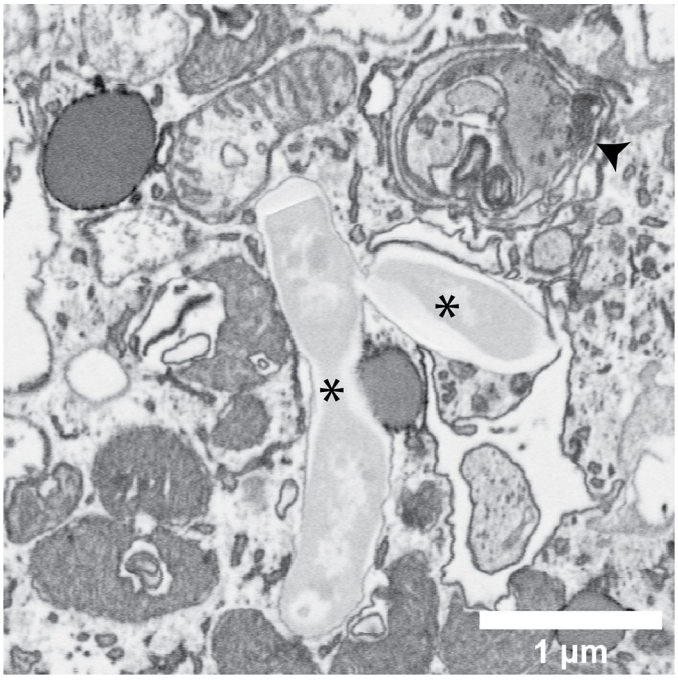

***Mycobacterium tuberculosis* is seen residing in the cytosol of a human macrophage by FIB SEM after successfully escaping from the autophagy machinery.** Bacteria are highlighted by asterisks (*). An autophagosome (arrowhead) containing vesicles and other membranes can also be seen.

What's next for you?

I am currently looking for postdoctoral opportunities in academia, continuing with cell biology of the endolysosomal/autophagy systems and diseases associated with them. I'd really like to continue with expanding my microscopy skills as well as building up new skills in biochemical approaches to understand the mechanism behind the observational biology.

Tell us something interesting about yourself that wouldn't be on your CV

As well as pursuing my PhD, I am a volunteer with St John Ambulance – a large first aid charity that normally provides first aid cover at events across the country. During the COVID-19 pandemic I have continued this volunteering by supporting the London Ambulance Service with additional ambulances to send to 999 calls.

Reference

Bernard, E. M., Fearns, A., Bussi, C., Santucci, P., Peddie, C. J., Lai, R. J., Collinson, L. M. and Gutierrez, M. G. (2021). *M. tuberculosis* infection of human iPSC-derived macrophages reveals complex membrane dynamics during xenophagy evasion. *J. Cell Sci.* **134**, jcs252973. doi:10.1242/jcs.252973