

## FIRST PERSON

# First person – Amelia Townley

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. Amelia Townley is first author on 'Mitochondrial survivin reduces oxidative phosphorylation in cancer cells by inhibiting mitophagy', published in JCS. Amelia conducted the research described in this article while a PhD student in the lab of Sally Wheatley at the University of Nottingham, UK. Her PhD research investigated how alterations to mitochondrial turnover benefit cancer cell progression and formation.

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

Survivin is a protein that is highly expressed in cancer. It not only enables cells to divide but also to escape cell death. In cancer cells, a small amount of the protein is found within the energy-producing organelles – the mitochondria. What survivin does when it is in the mitochondria, and how this might benefit cancer cells, is not fully understood. In this paper, we show that mitochondrial survivin prevents the removal of faulty mitochondria, causing an accumulation of defective organelles that are unable to produce much energy. Our data suggest that cancer cells compensate for this problem by switching their metabolism to glycolysis, a second mechanism of energy production that occurs in the cytoplasm, the thick solution that fills the centre of the cell. This switch has a range of advantages for cancer cells, as they often grow in harsh conditions such as low oxygen or with limited nutrients. This discovery improves our understanding of an additional role that survivin acquires in cancer cells – insight that may help devise new strategies to target survivin in cancer therapies.

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

The most challenging aspect of this project was an issue many biochemists face: protein purification. After months of optimisation experiments – changing incubation times, temperatures and growth medium composition – and a lot of perseverance, I was able to purify sufficient GST–Parkin to carry out an important experiment that helped me to determine where survivin functions within the mitophagic pathway. Another challenge was quantifying and analysing the data from hundreds of immunofluorescence images used throughout this paper. Luckily, other members of the lab were proficient in developing macros in Fiji, and they helped me to do this quickly and precisely.

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

For a long time we thought that survivin modulated mitochondrial metabolism in cancer cells by manipulating mitochondrial dynamics, as suggested in the literature. However, after discovering that survivin increased mitochondrial mass, we were



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disappointed to find that it did not regulate the fission and fusion machinery. Our 'eureka' moment then struck us – survivin must be modulating mitochondrial turnover. Suddenly it all became clear: a previous PhD student in the lab had found that survivin can limit autophagy. Because mitochondria are removed from the cell by a specialised form of autophagy, known as mitophagy, we revised our hypothesis to 'survivin inhibits mitophagy in cancer cells'. This defining moment set us on the right path that ultimately led to this paper. It really goes to show that if a result completely defies your original hypothesis, this shouldn't necessarily be disappointing; these moments can help you to re-evaluate the data and may ultimately result in a more logical outcome.

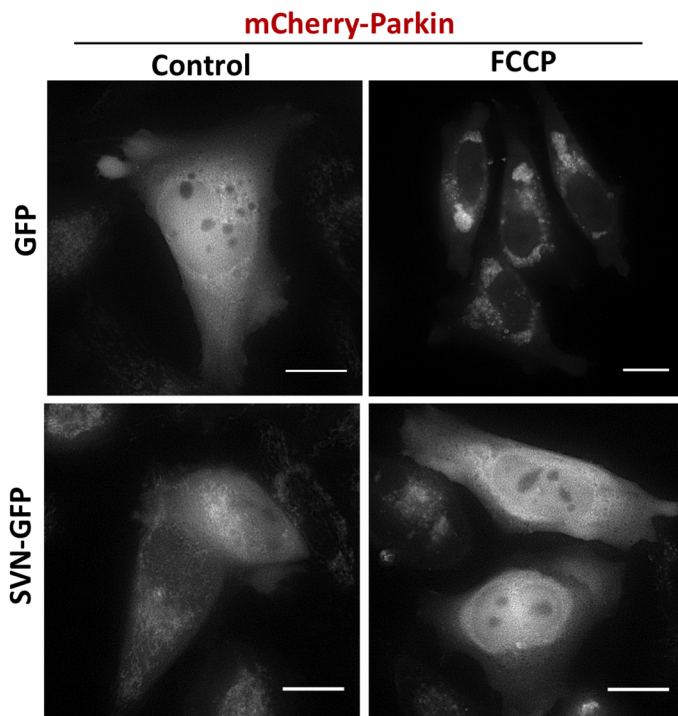
### Why did you choose Journal of Cell Science for your paper?

After attending Journal of Cell Science's 'Organelle–Cytoskeleton Interface' conference in Lisbon (in May 2019, when face-to-face conferences were safe!), I was taken aback by the broad audience that JCS reaches. As JCS is a highly respected journal that frequently publishes key, highly cited papers in this area, it was an easy choice to submit our paper to JCS.

### Have you had any significant mentors who have helped you beyond supervision in the lab? How was their guidance special?

My PhD supervisor's guidance throughout went above and beyond day-to-day experimental advice. With her direction and confidence in my work, I was able to overcome the 'imposter syndrome', which a lot of PhD students experience, and now I truly believe in my own capabilities.

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**Survivin prevents the mitochondrial translocation of Parkin.** HeLa cells expressing survivin-GFP (SVN-GFP) prevent the mitochondrial translocation of mCherry-Parkin post mitochondrial depolarisation and mitophagy stimulation (FCCP). Scale bars: 10  $\mu$ M.

### 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 think everyone who works in science can pinpoint the spark that initiated their curiosity, and for me it was due to the influence of one person. From a young age, my father, a practising geologist and micropaleontologist, would take me to beaches to collect fossils. Naturally, this led to an interest in biology, specifically evolution, which subsequently evolved into an interest in core cell biology processes, a topic I still find engaging and surprising.

One of the most interesting moments of my career so far was during my placement at 'Sense about Science', in which I found myself in parliament discussing scientific issues with MPs and science policy makers. There we helped to provide a platform for researchers to discuss their work directly with MPs, an experience that gave me a unique insight into how science policy decisions are made. I left with a greater appreciation of the need for greater dialogue between researchers and parliament (and I hope the MPs did too!).

### What's next for you?

I am about to submit my PhD thesis at the University of Nottingham, and have accepted a postdoctoral position at the Royal Veterinary College London, where I will continue to research mitochondria and their role in disease.

### Reference

Townley, A. R. and Wheatley, S. P. (2020). Mitochondrial survivin reduces oxidative phosphorylation in cancer cells by inhibiting mitophagy. *J. Cell Sci.* **133**, jcs247379. doi:10.1242/jcs.247379