

FIRST PERSON

First person – Imadeddin Hijazi

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. Imadeddin Hijazi is first author on 'Retrograde signaling mediates an adaptive survival response to endoplasmic reticulum stress in *Saccharomyces cerevisiae*', published in JCS. Imadeddin is an undergraduate research assistant in the lab of Amy Chang at the University of Michigan, Ann Arbor, MI, USA, investigating the mechanisms cells use to adapt to endoplasmic reticulum stress.

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

The endoplasmic reticulum (ER) is in charge of folding some of the proteins in the cell. This function is essential, so when the ER cannot properly fold proteins, it experiences stress. A lot of stress causes the cell to die. We have found that if the ER experiences a small amount of stress before experiencing a large amount of stress, the cells survive better (what we call 'adaptation'). In our paper, we found that when the ER is stressed, the mitochondria become stronger. Mitochondria create energy for the cell through a process called cellular respiration, which requires the electron transport chain to be active. Here, we show that when the ER is stressed, components of the electron transport chain increase. This increase is necessary for adaptation. The mitochondria use a process called 'retrograde signaling' to communicate their distress to the nucleus. Retrograde signaling is needed for the cells to adapt to ER stress.

“It was thrilling – despite the frustration – to realize we were on the right track early on, and that we were uncovering a previously un- or barely characterized response to ER stress.”

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

It was difficult to know where to look at times; many of these results were new to us, so figuring out our next step required some guesswork. To overcome this, we would look at various mutants using our handful of assays, and whatever stuck, we investigated further. It was thrilling – despite the frustration – to realize we were on the right track early on, and that we were uncovering a previously un- or barely characterized response to ER stress.

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

The Gcn4-lacZ data that showed the cells were not starving for amino acids when treated with tunicamycin. It's logical to take the data and assume there is a roundabout way that ER stress influences



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mitochondrial function. TORC1 inactivation and retrograde signaling have previously been shown to occur when cells were starving for amino acids, and it was reasonable to say that the amino acid transporters were simply stuck in the ER. However, this graph confirmed that our response was unique and independent of starvation.

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

We wanted a high-quality journal that would reach a wide audience of biologists.

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

Dr Amy Chang was an amazing mentor, encouraging me to look at the bigger picture in whatever I did, and helping me search for where my passions truly lie, even if they seem daunting. Jeff Knupp also played a tremendous role in guiding me toward choosing a research-heavy career.

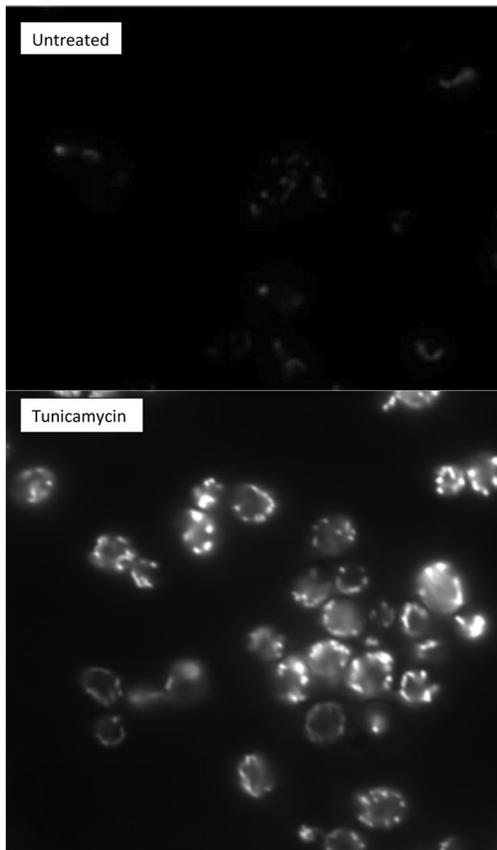
What motivated you to pursue a career in science, and what have been the most interesting moments on the path that led you to where you are now?

In high school, I enjoyed the curiosity that biology brought. How do these tiny units know their environment? How do they respond in a way that enhances their odds of survival? In college, I decided to pursue cell biology for my major, and I began working in Dr Chang's lab for the experience. I found research to satisfy – and frustrate – my curiosity.

Who are your role models in science? Why?

James Talmage, because despite what he wanted to believe and how easily he could have touted the archaeological finding as a

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Endoplasmic reticulum stress increases mitochondrial membrane potential in *Saccharomyces cerevisiae*.

confirmation of his faith, he chose to scrutinize the data until he found the truth. I think that's a good model for science; even if it goes against his or her best interest, it is a scientist's job to pursue and uncover the truth.

What's next for you?

I plan on applying to medical scientist training programs (and other MD/PhD programs) to pursue a career that incorporates research and clinical medicine, with the hopes of continuing basic science research.

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

I love stage managing and directing plays for my student theater organization, and I hope to get involved in community theater in the future.

Reference

Hijazi, I., Knupp, J. and Chang, A. (2020). Retrograde signaling mediates an adaptive survival response to endoplasmic reticulum stress in *Saccharomyces cerevisiae*. *J. Cell Sci.* **133**, 241539. doi:10.1242/jcs.241539