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FIRST PERSON

First person – Yixing Wu and Ying Bai

First Person is a series of interviews with the first authors of a selection of papers published in Biology Open, helping early-career researchers promote themselves alongside their papers. Yixing Wu and Ying Bai are co-first authors on 'Palmitoylated small GTPase ARL15 is translocated within Golgi network during adipogenesis', published in BiO. Yixing is a research fellow in the lab of Frances Wiseman at UCL Queen Square Institute of Neurology, London, UK, investigating Down's syndrome and Alzheimer's disease-related endo-lysosomal pathways and cathepsin deficits. Ying is a postdoc in the lab of Roger D. Cox at MRC Harwell Institute, Didcot, UK, investigating how fat cells are formed, and genes that are involved in regulating body fat distribution.

Yixing Wu and Ying Bai

What is your scientific background and the general focus of your lab?

Our respective backgrounds are neurobiology (Yixing Wu) and biochemistry (Ying Bai). We were working in Prof. Roger Cox's lab focusing on fat distribution and its role in regulating metabolic healthiness. The combination of expertise in biochemistry and cellular differentiation allowed us to study protein–protein interaction networks during fat cell development and enabled us to discover key posttranslational modifications of ARL15 to establish its unique subcellular localisation.

Yixing Wu is currently studying Down's syndrome and Alzheimer's disease-related mechanisms in Dr Frances Wiseman's group at UCL.

How would you explain the main findings of your paper to non-scientific family and friends?

In our body, fat tissue plays important roles in metabolism and energy storage as well as protecting our organs and keeping us warm. However, sometimes too much or too little fat tissue can cause harm. Fat tissue is made of fat cells. How a cell becomes a fat cell is still being investigated. Our paper studied a protein that may play a role in regulating the process of generating fat cells. We investigated its whereabouts within the cell during the process of maturation; we also determined which part of the protein allows it to stay there. We also found other proteins it may interact with. These findings deepen our understanding of the process of making fat cells.

What are the potential implications of these results for your field of research?

Our study showed the importance of posttranslational modification on cellular localization and revealed a dynamic morphological change that Golgi makes during adipogenesis, thus suggesting an important role Golgi plays during fat cell maturation process.

Yixing Wu's contact details: UCL Queen Square Institute of Neurology, Queen Square, London WC1N 3BG, UK.
Ying Bai's contact details: MRC Harwell Institute, Harwell Campus, Didcot, OX11 0RD, UK.
E-mail: yixing.wu@ucl.ac.uk; y.bai@har.mrc.ac.uk

What has surprised you the most while conducting your research?

ARL15's Golgi localization is palmitoylation-dependent, and it travels between different compartments of Golgi apparatus during adipogenesis.

What, in your opinion, are some of the greatest achievements in your field and how has this influenced your research?

I think some of the greatest achievements in the field are the discovery of the importance of fat distribution in determining metabolic healthiness and advances made in understanding the underlying genetic basis. Central obesity where fat is mainly stored around organs in addition to ectopic storage within organs such as pancreas, kidney and liver predisposes a person to type 2 diabetes, metabolic syndrome and cardiovascular diseases. However, fat that is stored around gluteal, subcutaneous and trunk regions of the body are less harmful because they serve as 'safe' lipid storage depots and help to store excess fat until required. Fat distribution is partly under the complex control of genes that may be differentially expressed between depots. Alleles of these genes influence whether fat is stored predominantly in subcutaneous or central depots. If subcutaneous fat has a reduced ability to store fat, excess fat accumulates in the central region and ultimately leads to lipid toxicity in other organs and insulin resistance. Our research interest is to find genes and mechanisms that are causal for insulin resistance due to insufficient fat storage in subcutaneous regions. We use the mouse as a model organism since they develop symptoms of type 2 diabetes or insulin resistance in a similar way to humans.

What changes do you think could improve the professional lives of early-career scientists?

Y.W.: I think there should be more career developing forums and platforms (either in person or online) for early-career scientists to share each other's thoughts and experiences. A platform to help early-career scientists to look for mentorship would also be beneficial.

Y.B.: I think there should be more long-term contracts for early-career scientists to establish meaningful and useful research topics,

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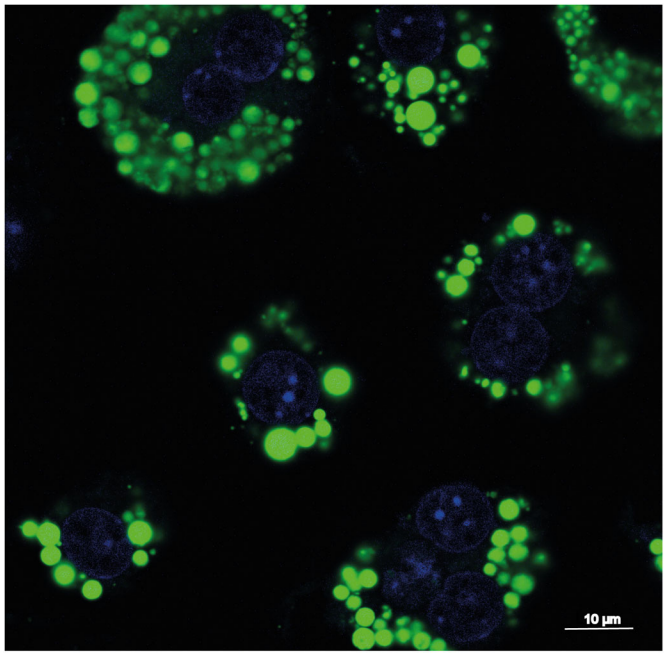

BODIPY staining of lipid droplet from differentiated 3T3-L1 mouse adipocytes.

which would allow them to follow up and write research grants with. Also, more mentors should be available for early-career scientists to approach given academia life requires lots of wisdom in project planning, budget spending, workload distribution, panel review defending, team building and networking.

What's next for you?

Y.W.: I am planning to stay in academia and continue studying Down's syndrome and Alzheimer's disease. I am going to apply for senior research fellowships in the future.

Y.B.: I am currently looking for postdoc positions in the research field of regenerative medicine, cancer or immunology. I like asking questions and design experiments to solve problems. I am a wet lab person and enjoy doing experiments, especially generating good high-quality data for robust research output. I would like to stay in academia, therefore applying for a senior research fellowship is what I would like to do in the future.

One thing you want to change for academic research?

Increase the length of contracts for postdoc positions.

Reference

Wu, Y., Bai, Y., McEwan, D. G., Bentley, L., Aravani, D. and Cox, R. D. (2020). Palmitoylated small GTPase ARL15 is translocated within Golgi network during adipogenesis. *Biology Open.* 10, bio058420. doi:10.1242/bio.058420