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FIRST PERSON

First person – Komaki Ninomiya

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. Komaki Ninomiya is first author on 'PLEKHG4B enables actin cytoskeletal remodeling during epithelial cell–cell junction formation', published in JCS. Komaki is a PhD student in the lab of Kazumasa Ohashi at Tohoku University, Sendai, Japan, investigating the mechanisms and spatiotemporal dynamics of cytoskeletal and cellular structures driven by Rho-GEFs.

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

The actin cytoskeleton is a complex network in the cells that drives various cellular behaviors. Our lab has been studying actin cytoskeletal dynamics, recently focusing on the Rho-GEF family, which is a central regulator of actin cytoskeletal remodeling. In our current paper, we have found that a Rho-GEF named PLEKHG4B is a novel player in epithelial cell–cell junction formation. In the initial stages of our study, we found that the cells exhibited an 'open' junction (which is thought to be an immature junction structure) when PLEKHG4B was depleted. Based on this observation, we explored further and finally demonstrated that PLEKHG4B plays a role in actin cytoskeletal remodeling in the late stage of cell–cell junction formation by regulating the contractility of actin filaments, which prompts the conversion from 'open' to 'closed' junctions. At the same time, we suggested that there is a cooperation between Rho-GEFs; PLEKHG4B regulates actomyosin by suppressing the activities of LARG and PDZ-RhoGEF, which are both other Rho-GEF family members.

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

When I started the current project as an undergraduate student, I did not know how to go ahead with my research even when I made interesting observations. In this sense, this paper itself is a unique challenge in my life. Thanks to the guidance of my supervisors, now I feel I have grown as a scientist.

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

One of the 'eureka' moments was the first time I saw the effect of the PLEKHG4B depletion on the cell–cell junction structure, in which actin filaments were split away from the cell–cell interface (which we later refer to as an 'open' junction). This observation provided me with an exciting hypothesis and a new idea to uncover the role of PLEKHG4B.

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

I wanted our paper to reach a large audience who would be interested in our findings. Additionally, I have always been fascinated by JCS papers.

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Komaki Ninomiya

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

Although there are too many to name, the mentorship of both my supervisors, as well as other senior members of my lab, has been essential for my PhD training. One of the wonderful things I learned from them is that in science, nothing can be taken for granted. They emphasized the importance of questioning the result of every experiment, as well as enjoying the experiment itself.

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 previously wanted to study literature, as I enjoyed the experience every time I read a new book. However, that all changed when I first looked down a microscope and saw the structure of the cytoskeleton for the first time. At that moment, I found myself eager to see and learn more about the elaborate mechanisms behind the cytoskeletal structure. I believe it was the beauty of the cytoskeletal structure that led me to where I am today.

Who are your role models in science? Why?

My supervisors, Professors Kensaku Mizuno and Kazumasa Ohashi, helped me develop a passion for cell biology. They have been inspiring me since I first began working with them.

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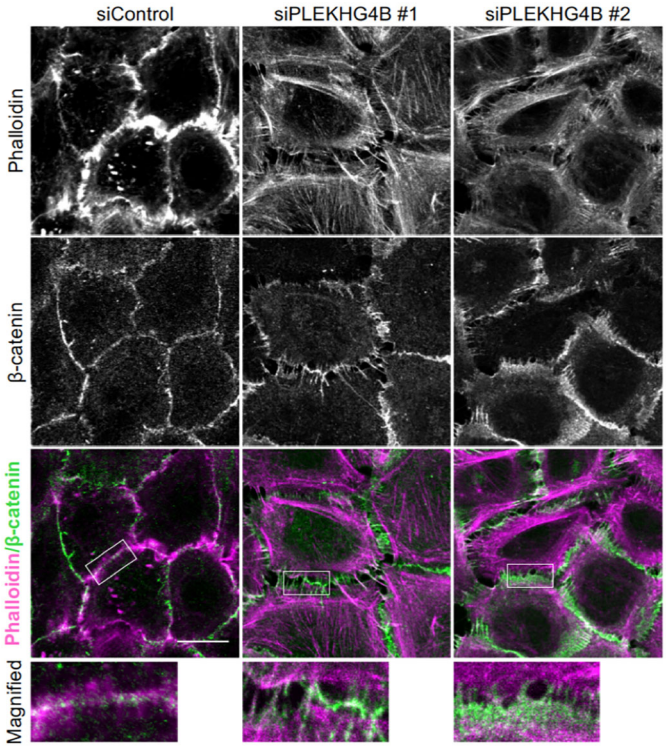

PLEKHG4B depletion delays cell–cell junction formation. A549 cells were treated with siRNAs targeting PLEKHG4B and analyzed by staining for actin (phalloidin, magenta) and adherens junctions (β-catenin, green). Bottom panels show magnified images of the regions marked by white boxes. PLEKHG4B-depleted cells show 'open' junctions, whereas control cells show 'closed' junctions.

What's next for you?

I would like to continue researching the mechanisms underlying cytoskeleton behavior and to expand my research to other model organism systems in addition to culture cells.

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

I enjoy cooking by myself, eating at new restaurants, and spending quality time with my friends, family and colleagues. This allows me to remain relaxed and enjoy every day.

Reference

Ninomiya, K., Ohta, K., Yamashita, K., Mizuno, K. and Ohashi, K. (2021). PLEKHG4B enables actin cytoskeletal remodeling during epithelial cell–cell junction formation. *J. Cell Sci.* **134**, jcs249078. doi:10.1242/jcs.249078