

## FIRST PERSON

## SPECIAL ISSUE: CELL BIOLOGY OF THE IMMUNE SYSTEM

# First person – Jiarui Bi

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. Jiarui Bi is first author on 'Epidermal growth factor receptor signaling suppresses  $\alpha\text{v}\beta 6$  integrin and promotes periodontal inflammation and bone loss', published in JCS. Jiarui is a PhD candidate and lab associate in the lab of Hannu S. Larjava at the University of British Columbia, Vancouver, Canada, investigating new therapies for periodontal disease by soft tissue generation and interruption of biofilm inducement of the disease.

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

About half of the adult population suffers from periodontal (gum) diseases, which can result in gum inflammation, loss of teeth, and even potentially contribute to many systemic diseases. In periodontal diseases, the junctional epithelium that connects the gingiva to the tooth enamel is injured by multispecies bacterial biofilms. It transforms to the pocket epithelium that forms a periodontal pocket between the gingiva and teeth. Gingival inflammation and bone loss around the teeth will follow. The  $\alpha\text{v}\beta 6$  integrin presented in junctional epithelium maintains anti-inflammatory transforming growth factor- $\beta 1$  (TGF- $\beta 1$ ) signaling and periodontal health. However, the expression of  $\alpha\text{v}\beta 6$  integrin is significantly downregulated in the pocket epithelium during periodontal diseases. In this study, we show that the bacterial biofilms can suppress the expression of  $\beta 6$  integrin mRNA and protein in cultured gingival epithelial cells (GECs) by attenuating TGF- $\beta 1$  signaling through autocrine GEC EGFR signaling. This pathologic process leads the pro-inflammatory response in GECs. We also show that the biofilm-initiated  $\beta 6$  integrin downregulation in GEC can be prevented by blocking EGFR signaling which could serve as a novel approach to reduce inflammation and bone loss in periodontal disease.

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

One of the challenges we met in this study was the establishing of mouse-based experimental periodontitis model by the silk ligature technique. The size and structure of mouse oral cavity are relatively small. We needed to tie the 6–10 silk sutures around the super tiny second molar of mouse maxillary with tweezers with the minimum damage to the mouse gum tissue. The whole procedure needed to be performed quickly under anesthesia. Moreover, we needed to change the drug-soaked silk sutures for the different local delivery groups every other day for 2 weeks. The experiments needed patience, experience and excellent manual skill in this animal model. We overcame these challenges through practice and hundreds of attempts. In the end, we induced periodontal bone loss and inflammation in 2 weeks and were able to prevent most of it with EGFR inhibition.



Jiarui Bi

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

Of course. It has happened many times in my research. In the present study, it is the first time that we show that this integrin located in specific dental epithelium plays a significant role in regulation of periodontal inflammation and subsequent bone loss. It provides a new paradigm for tissue destruction in periodontal disease. Thus, when we discovered these novel findings, I was excited. These are the moments that motivate me to stay in the academic field. I hope that we can further explore this research and translate our findings into patient care that could help millions of patients worldwide.

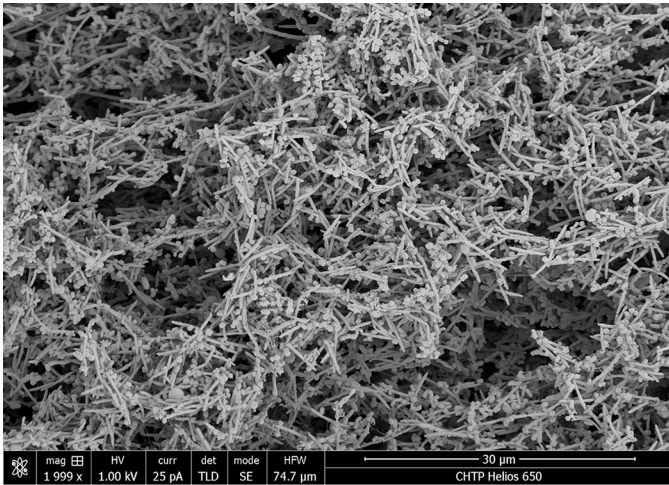
### Why did you choose Journal of Cell Science for your paper?

The Journal of Cell Science is a high-quality journal of cell biology with a great reputation. We chose JCS because we would like our work to reach a broad audience in the field.

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

I would like to extend my deep appreciation and gratitude to all the technical staff members working at the Lab of Periodontal Biology at UBC, in particular Dr Leeni Koivisto for her kind advice and assistance throughout my PhD study. Her work is always detailed and methodical. She always comes out with bright ideas and points out key points in the study. Her tireless passion for research makes her a strong role model that influences the entire lab. Without her, my studies would not progress this smoothly and successfully.

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Structure of three-week cultured oral bacterial biofilm from a donor with periodontitis.

### Who are your role models in science? Why?

I want to say Elon Musk is my role model. Some might say that he is not a scientist. But what I admire is that he has dreams and he takes

action to accomplish these dreams, which contributes to society and even the progress of humanity. And I think that's what most scientists, including me, are trying and doing. Also I respect all the people who are dedicated to science, as I understand that research work is never easy.

### What's next for you?

I am finishing my PhD studies, and plan to graduate early next year. I am passionate about research. Now, I am looking for a post-doc position to continue my academic journey.

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

In my life, I am a big Lego fan and a PC builder. I am also addicted to many simulation games, like city building, etc. Furthermore, I do home repair and upgrades. So basically, I like to build stuff.

### Reference

Bi, J., Koivisto, L., Dai, J., Zhuang, D., Jiang, G., Larjava, M., Shen, Y., Bi, L., Liu, F., Haapasalo, M. et al. (2020). Epidermal growth factor receptor signaling suppresses  $\alpha\text{v}\beta 6$  integrin and promotes periodontal inflammation and bone loss. *J. Cell Sci.* **133**, 236588. doi:10.1242/jcs.236588