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FIRST PERSON

First person – Chaitali Khan

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. Chaitali Khan is the first author on 'The initiator caspase Dronc plays a non-apoptotic role in promoting DNA damage signalling in *D. melanogaster*', published in Journal of Cell Science. Chaitali is a PhD student in the lab of Basuthkar J. Rao at the Tata Institute of Fundamental Research, Mumbai, India, investigating the principles of tissue homeostasis in the context of DNA damage, cell death and cellular signalling.

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

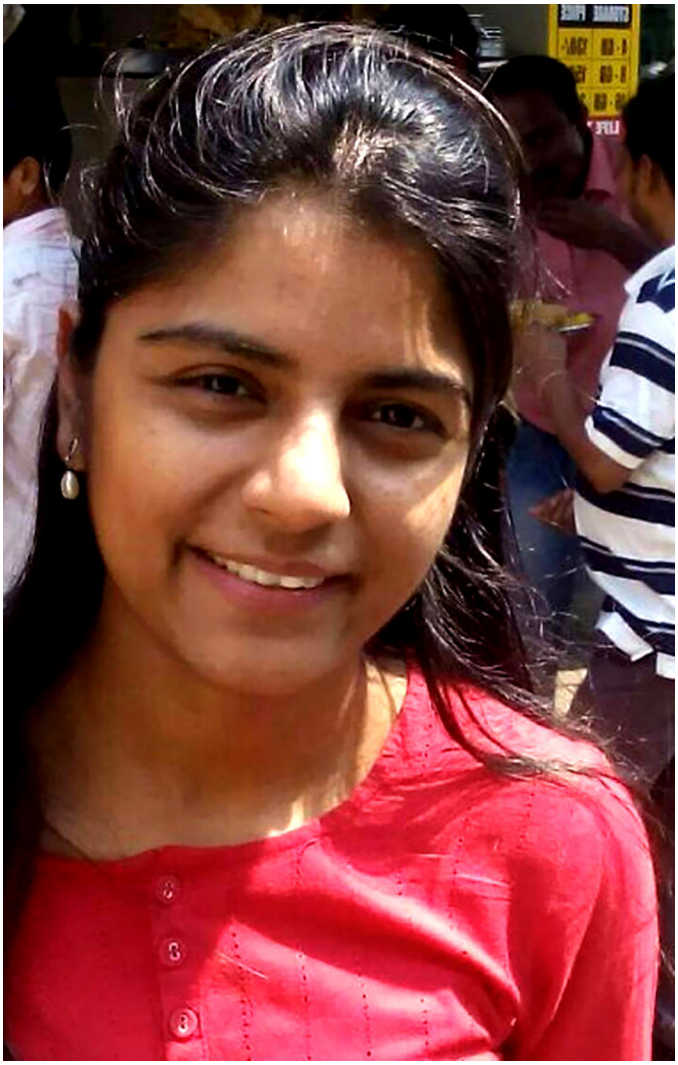
Our genome is prone to damage during its replication, as a result of exposure to genotoxins, or even because of sunbathing. Inside a cell, this damage is kept under check by DNA repair machinery. Unrepaired DNA damage often leads to the progression of cancer or cellular malfunctioning; to overcome this, damaged cells are removed from the system by the process of cell death. However, premature cell death can also lead to defects in a tissue. So how does a cell decide whether to repair or to die? My work focuses on the role of the cell death machinery in promoting DNA repair when levels of damage are low and there is still a chance to repair the DNA. Specifically, our paper implicates cell death caspases in this decision-making task before a cell undergoes death.

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

Very few groups are working on DNA repair in *Drosophila*, so there were lot of challenges in terms of proper reagents such as antibodies and *Drosophila* stocks. It was difficult to determine whether γH2av was arising as a result of DNA damage signalling or cell death. However, because genetics assays can be performed in *Drosophila* quickly and elegantly, they helped me overcome these problems, and we clearly dissected the DNA damage response from the cell-death-associated γH2av response.

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

In the beginning, it was a simple observation that suppressing the initiator caspase Dronc leads to a reduction in the γH2av response. I remember the moment when two of my experiments consolidated the non-apoptotic role of Dronc in DNA damage signalling. These were the suppression of effector caspases leading to an increase in the γH2av response during DNA damage, and the absence of the γH2av response during cell death induction. These were the two contrasting results that made me believe in my study. I remember running out of the confocal room and explaining it to my PI Prof. B. J. Rao, and making a call to my friend (and co-author on the paper) Sonia Muliyil to discuss what I observed.



Chaitali Khan

" …research is nothing more than my little experiments in the backyard that I used to do with my brother"

Have you had any significant mentors, and how have they helped you?

Yes, I have many of them. When I was young I was not interested in research, but I loved to experiment. My brother was also a child geek, and I enjoyed our little backyard science experiments and vivid explanations about the phenomena occurring in nature. I think those were the events that shaped my scientific reasoning and appreciation. Another significant mentor was my science teacher in

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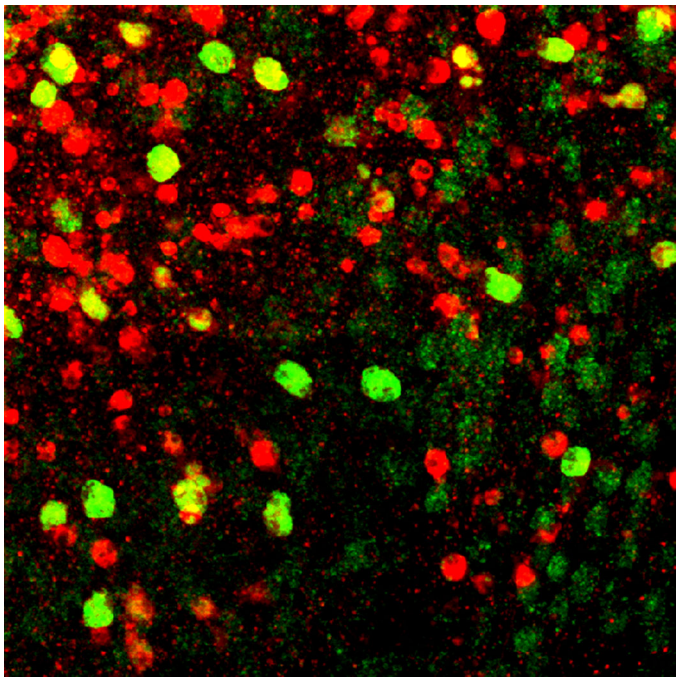

γH2av (green) and cleaved caspase 3 (red) in the *D. melanogaster* wing imaginal disc.

my high school. She helped me realize the joy of understanding things, and that research is nothing more than my little experiments in the backyard that I used to do with my brother.

What's the most important piece of advice you would give first-year PhD students?

Keep yourself open to multiple possibilities and do not predict the outcome of an experiment. My work, especially this story, is a good example. I started with a single observation that Dronc suppression leads to a reduction in the γH2av response, and ended up with an unconventional function of a caspase in promoting DNA repair instead of cell death. I would say many a time we discard our results because they are not in accordance with what we were expecting, but most of the time these are the most exciting results and lead to new discoveries.

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

I believe having a good start-up grant to establish a firm footing during the early stages of a career is of immense help. Also, we should be evaluated on the content of the work instead of the impact factor of the journals in which one's papers are published. This will help early-career scientists to pursue their science with passion and uncover interesting phenomena, which I think gets limited these days as a result of high-pressure, impact-factor-driven publishing criteria.

"Keep yourself open to multiple possibilities and do not predict the outcome of an experiment"

What's next for you?

I will be moving on to pursue a postdoc with Prof. Nicholas Baker at the Albert Einstein College of Medicine, New York, USA, where I will be studying the various mechanisms governing cell completion in minute mutants. I hope for another exciting journey and the understanding of newer problems, especially the function of the ribosomal machinery in cellular homeostasis.

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

Apart from my regular lab work, I spend time in teaching college students to spread the excitement and joy of research. I enjoy reading fiction, and one of my all-time favourites is Harry Potter by J. K. Rowling. I have been following the Harry Potter series since high school. I believe it has had a huge impact in shaping me as a person, and one of my dreams is to meet J. K. Rowling in person.

Reference

Khan, C., Muliyil, S., Ayyub, C. and Rao, B. J. (2017). The initiator caspase Dronc plays a non-apoptotic role in promoting DNA damage signalling in D. melanogaster. *J. Cell Sci.* **130**, 2984-2995.