

Free, publicly available health data proves to be research gold mine

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From left: Pinaki Bose and Lubaba (Aurna) Khan sourced big data to try to explain why some cancer patients respond to immunotherapy while others do not. The findings are published in *Nature Communications*. Credit: Riley Brandt, University of Calgary

It's a popular question: What did you do over the summer? For Lubaba



(Aurna) Khan, the summer of 2018 will be one she will never forget.

It started on a high note and ended on an even higher one. In June, Khan walked across the stage to accept her degree as a University of Calgary Bachelor of Health Sciences graduate. She received an O'Brien Centre Summer Studentships award, landing a research job with Dr. Pinaki Bose, Ph.D. Then, unexpectedly, she made a discovery that could help cancer patients throughout the world.

Khan spent the summer hunched over a computer interpreting free, publicly accessible health and genomics data. "Bioinformatics is the new frontier of medical science," says Khan. "I started my <u>medical education</u> as a biomedical student, looking at cells through a microscope, but looking at <u>cancer biology</u> with the assistance of a computer opens up a new way of thinking about research."

Khan was comparing the differences between genes found in <u>cancer</u> <u>cells</u> and those found in normal cells. Working under the guidance of Bose, Khan learned how to construct questions and hypotheses. It was a bumpy start.

"We wanted to find out how immune genes in cancer cells might be associated with <u>immunotherapy</u> response. There was nothing there, and we were disappointed," says Bose, who is an adjunct assistant professor in the departments of biochemistry and <u>molecular biology</u>, and oncology, and is a member of the Arnie Charbonneau Cancer Institute at the Cumming School of Medicine.

"We adjusted our focus, and discovered that another set of genes, those associated with the extracellular matrix, had a direct connection to how <u>cancer patients</u> respond to immunotherapy."

Immunotherapy has become a popular treatment for some cancers. It



uses the body's own immune system to attack and kill cancer cells. The treatment is effective, but only for a select few, around 20 to 30 per cent of patients. There is not a clear understanding as to why some people respond better to immunotherapy than others.

The extracellular matrix may hold some of the answers; it grows within and around both healthy and cancerous cells.

"We found that genes associated with the <u>extracellular matrix</u> are overly produced in patients who do not respond to immunotherapy," says Bose, who is also the director of translational research for the Ohlson Research Initiative. "These <u>genes</u> are produced by cells surrounding the tumour, and they form a barrier which helps cancer <u>cells</u> evade detection by the immune system."

The UCalgary team shared the discovery with researchers at the University of Toronto who have validated the findings, now published in *Nature Communications*. "I was like, wow, I'm a student. I didn't realize how important this was until Dr. Bose pointed out how significant this discovery could be in <u>cancer</u> treatment. It kind of threw me off, but I was extremely excited at the same time," says Khan, who is a co-first author on the study.

The researchers looked more deeply at their findings and discovered a gene signature, which could lead to the development of a simple test that could help determine which patients would benefit from immunotherapy. The next step will be to prove the findings in a lab.

"Analyzing <u>big data</u> won't replace bench science, but it a great tool for hypothesis-building and leading to new insights," says Bose.

For Khan, this experience has changed the course of her future studies. She always planned to pursue a master's degree in public health, and now



sees a future focused on data analysis rather than data collection.

More information: TGF- β -associated extracellular matrix genes link cancer-associated fibroblasts to immune evasion and immunotherapy failure, *Nature Communications* (2018). DOI: 10.5281/zenodo.1410639, www.nature.com/articles/s41467-018-06654-8

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