

Editorial

Nanobytes

Radhika Vaishnav

Department of Molecular Informatics, Natureka Life Sciences, Vadodara, Gujarat, India.



***Corresponding author:**

Radhika Vaishnav,
Department of Molecular Informatics, Natureka Life Sciences, Alkapuri, Vadodara - 390 007, Gujarat, India.

radhikavaishnav@gmail.com

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BENCH TO BEDSIDE BYTE: GLIOBLASTOMAS UNRAVELED WITH TRANSLATIONAL RESEARCH

A recent study in Cell^[1] exemplifies the elegance of utilizing new technology and data mining tools to understand a complex disease. International collaborators studied glioblastoma tumors at the single-cell level. Glioblastomas were already known to be heterogeneous in cell type, and that the tumors could change their phenotype over the course of time. The authors of this study performed single-cell RNA sequencing on more than 24,000 glioblastoma cells from 20 adult and eight pediatric patients. The researchers describe their discovery of four different types of cells, each with its own distinct gene expression profile, of which 15% of the cells exhibited two genetic programs simultaneously. This was suggestive of the cells undergoing a switch from one form to another. No translational research is complete without going back and forth between clinic and bench. The researchers injected human glioblastoma cells into mice, a single subtype into a single mouse. The mice developed tumors that were heterogeneous – containing not just the tumor subtype that was injected, but a mixture. They clearly proved that a switching can and does occur between subtypes of glioblastoma cells. This finding has great implications in understanding and treating this complex disease.

TARGETED THERAPY BYTE: POLO TRIAL

Advanced pancreatic cancer patients with germline BRCA1 or BRCA2 gene mutations may benefit from a targeted drug used to treat ovarian and breast cancers. The trial, known as POLO, involved administration of PARP inhibitor olaparib (Lynparza) post-chemotherapy, in which out of 92 patients, 18 showed partial response. These preliminary results were published in NEJM recently.^[2] However, the patients did not show any improvement in overall survival. Thus, there is much scope for further work in this area, such as combinatorial approach with immunotherapy.

BASIC SCIENCE BYTE: TUMOR NECROPTOSIS IMPROVES IMMUNOTHERAPY

Necroptosis is a type of controlled necrotic cell death that is caspase-independent while it is dependent on receptor-interacting serine-threonine protein kinase 1 (RIPK1) and RIPK3 activity. In a study published in the journal Science Immunology,^[3] researchers delivered genes for the proteins that drive necroptosis into tumor cells, then injected the tumor cells into mice. They did the same thing with normal cells. The researchers saw that in combination with immunotherapy, there was tumor regression and extension of life span in the rodents. Much work is needed

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going forth on understanding the inflammatory signals of necroptotic cells and how they may differ in different cancers and cell/tissue types.

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