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GLUCOSE ADDICTION CAN FOMENT TUMOURS

Relevant for: Science & Technology | Topic: Science and Technology- developments and their applications and effects in everyday life

Genes at play: One gene promotes glucose uptake and consumption by breast cancer cells whereas another does the opposite. | Photo Credit: Vasyl Dolmatov

Breast cancer is the top cause of cancer-related deaths in Indian women and is the most prevalent type of cancer in women. Worldwide statistics show that about two million new cases were detected in 2018.

Researchers studying cancer metabolism have often noted that tumour cells are addicted to glucose. Once glucose enters the cell, it quickly gets fed to pathways and is utilised to multiply and grow. In contrast, normal cells primarily use glucose for energy production. This reprogramming of glucose metabolism by cancer cells often alters the response to drugs used in cancer treatment.

An international team has discovered the conflicting roles of two genes in regulating glucose utilisation by breast cancer cells. They revealed that the CBX2 gene promotes glucose uptake and consumption by breast cancer cells whereas CBX7, a sister gene, does the opposite. Targeting these genes and/or their glucose pathway can pave way for new cancer treatments.

“The reason we chose to study CBX genes is because of their essential role in human embryo development. Since metabolic requirements during embryogenesis and carcinogenesis share a striking similarity, we got interested in CBX genes and investigated their role in breast cancer,” says Mohammad Askandar Iqbal from Jamia Millia Islamia University who led the study.

The researchers identified the roles of the two genes by studying molecular data from over 3,000 breast tumour samples. In normal breast cells, they found lower CBX2 but higher levels of CBX7. In breast cancer patients they noted the opposite – higher CBX2 and lower CBX7 expression. Among breast cancer patients, those with higher CBX2 and lower CBX7 expression showed reduced survival, the study published in *Molecular Oncology* reported.

When asked if gene therapy – where one can disrupt the problematic gene – can be used here, Dr. Iqbal explains: “We can try to specifically knock out the CBX2 gene in breast tumour cells; however, we cannot take the chance of knocking the gene out of our body as it plays an essential role in human embryonic development. Alternatively, we can target the glucose consumption by breast cancer cells.”

The team also performed a drug sensitivity analysis to find out which FDA-approved drugs could be used to treat breast tumours with higher CBX2 and/or lower CBX7 expression levels. They identified methotrexate and rapamycin as candidate drugs to which patients with higher CBX2 expression in breast tumours may respond favourably.

“To further enrich our knowledge of therapeutic biomarkers, we are interested in delving into the mechanistic basis of these important observations and also profile the dependency of breast tumours on this pathway for designing personalised treatment strategies,” adds Dr. Iqbal.

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