

Novel regulatory mechanism for cell division found

November 21 2014

A protein kinase or enzyme known as PKM2 has proven to control cell division, potentially providing a molecular basis for tumor diagnosis and treatment.

A study, led by Zhimin Lu, M.D., Ph.D., professor of neuro-oncology at The University of Texas MD Anderson Cancer Center, showcased the non-metabolic abilities of PKM2 (pyruvate kinase M2) in promoting tumor cell proliferation when cells produce more of the enzyme.

The study results were published in today's issue of *Nature Communications*.

Dr. Lu's group previously demonstrated that PKM2 controls gene expression by binding to transcriptional factors and phosphorylating histone, proteins that have the unique ability to turn genes on and off. Phosphorylation is a process by which a phosphate group is added to a protein.

"PKM2 is expressed at high levels during [tumor progression](#) and is important for cell growth. However there's been little information about whether it directly controls cell division." said Lu. "Our findings underscored its function in [tumor formation](#) during the final stages of cell division known as cytokinesis."

Understanding how cytokinesis goes awry is important since [abnormal cell division](#) impacts tumor cell growth and spread. Lu's team looked at

the role of PKM2 in brain tumor development in mice. After analyzing the protein-coding gene, MLC2 (myosin light chain 2), Lu's group revealed how phosphorylation of MLC2 by PKM2 in [brain tumors](#) occurs. Phosphorylation of MLC2 controls a process which allows separation of a dividing parental cell into two 'daughter' cells.

"The results revealed that PKM2-regulated MLC2 phosphorylation and the related cytokinesis are instrumental in brain tumor development and are found to precisely control [cell division](#)," said Lu. "More importantly, our research shows that PKM2-regulated cytokinesis occurs in malignant tumors with bad outcome, such as glioblastoma, pancreatic cancer, and melanoma."

Tumor cells, in which certain protein-coding genes (EGFR, K-Ras and B-Raf) are activated, develop new patterns of "molecular signatures" for regulating cell proliferation. These changes enable the [tumor cells](#) to coordinate their metabolism and cycle progression through PKM2.

Provided by University of Texas M. D. Anderson Cancer Center

Citation: Novel regulatory mechanism for cell division found (2014, November 21) retrieved 27 April 2024 from

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