Commentary

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Recognizing a New System to Safeguard from Glioblastoma

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Description

A group of people have identified a protein called RanBP6 as a new regulator of Epidermal Growth Factor Receptor (EGFR). It was shown how silencing of RanBP6 promoted glioma growth, by up-regulating EGFR expression. Moreover, reconstitution of RanBP6 in a mouse xenograft model leads to reduction in tumor growth. These findings might have important clinical implications.

Malignant brain tumors represent about 3% of the known cancers and every year about 100,000 new cases are diagnosed worldwide. Glioblastoma is an aggressive type of cancer that can occur in the brain or spinal cord. Glioblastoma forms from cells called astrocytes that support nerve cells. Glioblastoma can occur at any age, but tends to occur more often in older adults. It can cause worsening headaches, nausea, vomiting and seizures. Glioblastoma is the most common and lethal primary central nervous system tumour. A decade of studies has underlined the complexity of the glioma genome, however, the functional significance of the vast majority of the genetic alterations remains elusive.

The Epidermal Growth Factor Receptor (EGFR) is a transmembrane protein that is a receptor for members of the epidermal growth factor family of extracellular protein ligands. The epidermal growth factor receptor (EGFR) plays a critical role in normal development and in human cancer. EGFR is one of the first receptor tyrosine kinases linked to human cancer and represents an important drug target in oncology. Aberrant activation of EGFR in cancer stimulates tumour growth and is primarily attributed to increased gene copy numbers or gain-of-function mutations. However, it can also result from defects in EGFR feedback regulation.

Some people have discovered a novel layer of complexity in the regulation of EGFR and also identified that previously uncharacterized protein RanBP6, is a modulator of EGFR expression. It was shown that RanBP6 is an importin family member that regulates the nuclear import of signal transducer and activator of transcription 3 (STAT3). RanBP6 silencing impairs STAT3 nuclear translocation, leading to transcriptional depression of EGFR and increased EGFR pathway output.

A study showed for the first time that STAT3 is a direct inhibitor of EGFR expression. STAT3 inhibitors are currently being investigated for the treatment of glioblastoma and other tumor types. Inhibiting STAT3 signaling could lead to an undesired activation of EGFR signaling.

Focal deletions of the RanBP6 locus were found in a subset of glioblastoma patients and silencing of RanBP6 promoted glioma growth in glioma mouse model, by up-regulating EGFR expression. Moreover, reconstitution of RanBP6 in human glioma cell lines that lack its expression lead to reduction in tumor growth in a xenograft mouse model.

The results provide an example of EGFR deregulation in cancer through silencing of components of the nuclear import pathway. They have identified a new link between the Ran-GTPase nuclear transport pathway and key cancer signaling pathways which warrant further study as inhibitors targeting nuclear transporters enter clinical evaluation as cancer.