OAKLAND UNIVERSITY

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OU Research Team Studies Brain Tumors

Brain tumors result from abnormal and uncontrolled cell division. A **glioma** is a particularly aggressive and invasive type of tumor. **Hypoxia**--the lack of oxygen--causes a glioma to be even more aggressive, and to be resistant to **chemotherapy** and **radiation**. The molecule **A Disintegrin and Metalloproteinase-17** (ADAM17) enhances the production of many **growth factors**, and therefore drugs that inhibit ADAM17 may be useful in treating gliomas and may improve patient survival. Hypoxia induces expression of ADAM17 and increases the invasiveness of a glioma. Another molecule, **Specificity Transcription Protein-1** (Sp1) may play a role in the **transcription** of ADAM17. Therefore, one hypothesis is that Sp1 controls glioma invasivemess under hypoxic conditions.

A team of researchers, funded by the **National Institutes of Health** and led by Distinguished Professor **Michael Chopp** of the **Department of Physics**, tested this hypothesis in their recent paper **Transcription Factor Sp1 Induces ADAM17 and Contributes to Tumor Cell Invasiveness Under Hypoxia** (**Journal of Experimental & Clinical Cancer Research**, Volume 28, Article Number 129). The team includes OU graduate student and lead author Alexandra Szalad and **Biomedical Sciences: Medical Physics** alumnus Mark Katakowski. They conclude that "our findings suggest the Sp1 transcription factor mediates ADAM17 expression under hypoxia, regulates glioma invasiveness, and thus, may be a target for anti-invasion therapies."

SUMMARY
Does Sp1 control tumor invasiveness under hypoxia?

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