JILLANN MAYLE, ALICE MAGRO, and ALBERT MAGRO, Department of Biology, Fairmont State University, Fairmont, WV, 26554. **Processing of cellular proteins by activated matriptases and metalloproteinases in apoptotic glioblastoma cells.**

Apoptosis is a major mechanism of cell death that occurs in the chemotherapeutic and the radiological treatment of malignant tumors. Glioblastoma is a type of brain cancer that does not readily metastasize, but is very invasive and highly malignant. Chemotherapeutic regimens for glioblastomas and other high grade gliomas have fallen short of providing effective treatment. Clinical studies comparing chemotherapeutic agents have indicated increased tumor shrinkage and a very slight increase in median survival times, but no evidence for an increase in survival rates. Apoptotic cells have characteristics that are different from non-apoptotic cells. Our in vitro studies demonstrate that LN18 glioblastoma cells have a significant increase in the cell surface metalloproteinase (ADAMs 10 and 12) and serine-protease (matriptase) activities as the cells proceed through apoptosis. Flow cytometry and ELISA data show that class I histocompatibility antigens (HLA-ABC), along with the activated complement component C3b are degraded as the glioblastoma cells proceed through apoptosis. Histocompatibility and complement components are important constituents in adaptive and innate immune defense systems. The ability of apoptotic glioblastoma cells to degrade these constituents could be relevant to the propensity of glioblastomas to escape immune surveillance. Conversion of zymogenic MMPs and matriptases could also be relevant to their ability to transform pro-TGFβs and other immuno-suppressive molecules into their active forms. Although apoptosis is the prominent pathway of cell death in the treatment of tumors, there is the possibility that cells in varying stages of apoptosis could promote heretofore unrecognized immunosuppressive consequences.