Improved therapies for high grade glioma (HGG) are imperative, as the median survival after diagnosis with grade IV glioma is 15 months. Recently pathways regulating neural development, such as the bone morphogenetic protein (BMP) pathway, have been investigated as potential therapeutic targets in HGG. In xenograft transplant models, BMP signaling has been shown to have a tumor suppressive effect on the subpopulation of cells known as glioma-initiating or glioma stem cells. However, the degree to which BMP signaling plays a role in the bulk tumor cells or the more differentiated component in HGG is unknown. To investigate these questions we used both human samples of HGG and a novel murine model of human HGG established in our laboratory. We determined that BMP signaling is present and active in human HGG using a human HGG tissue-microarray. Our analysis of phospho-smad1/5/8 staining suggests that BMP signaling is present and active in most cells in many HGG gliomas, suggesting that BMP signaling is not limited to the glioma stem cell compartment. To examine the role of BMP signaling in differentiated, tumorigenic astrocytes, we deleted the BMP type IA receptor gene (Bmpr1a) in transformed astrocytes, effectively abrogating canonical BMP signaling in these cells. The cells were then transplanted orthotopically into immunocompetent adult host mice. Preliminary data suggest that, while BMP signaling may be tumor suppressive in stem-like cells, it acts as a tumor promoter in differentiated tumorigenic astrocytes. Compared to controls receiving cells with intact Bmpr1a, mice receiving Bmpr1a-knockout cells showed a significant increase in survival time upon orthotopic injection (21 vs. 52 days, \( p = 0.001 \)). In vitro, deletion of Bmpr1a in oncogenic astrocytes resulted in decreased proliferation and increased Olig2 expression. Studies to further investigate the role of BMPs in HGG are underway.