Programmed Cell Death – 1 (PD1) inhibition activates tumorspecific T-lymphocytes and is an effective clinical therapy against some cancers. Preclinical data regarding immune checkpoint inhibitors against malignant glioma is scant, and interim analyses of clinical trials suggest modest effect in patients as single agents. We examined PD-1 inhibition in murine glioblastoma models in combination with other immunomodulatory agents. Methods – Syngeneic glioma tumors (GL261 and CT2A) were implanted intracranially in C57/B6 mice. In separate experiments, PD-1 inhibition was combined with antibody blockade of T-cell immunoglobulin and mucin protein 3 (TIM3), ligation of OX40 on T-lymphocytes, or vaccination with irradiated GM-CSF expressing tumor cells. Systemic antitumor immunity and tumor infiltrating lymphocytes were analyzed by ELISPOT assay and flow cytometry, respectively. Results - In both syngeneic glioma models, day 3, 6, and 9 systemic delivery of a monoclonal antibody against PD-1 led to increased survival vs. controls. In animals with GL261 intracranial tumors, survival was improved by combination of PD-1 blockade with subcutaneous injection of irradiated GM-CSF expressing GL261 tumor cells, with antibody blockade of T-cell immunoglobulin and mucin protein 3 (TIM3), or binding of OX40 on T-lymphocytes by an activating antibody. In most cases, ELISPOT analyses demonstrated enhanced Th1 immunity by combination immunotherapies. Vaccination was associated with an increased intratumoral CD8+ T lymphocyte / FoxP3+ T lymphocyte ratio. Conclusion –Blockade of PD-1 on T lymphocytes in glioma-bearing mice is active. Both antitumor immunity and survival can be enhanced by combination of PD-1 inhibition with agents that activate antitumor immunity by complementary mechanisms.