Supplemental legends

SFigure 1. DEX-treatment decreases proliferation but does not enhance glioma cell death. A) Representative immunohistochemical staining for Ki67 in vehicle and DEX-treated mice. Corresponding bar graph shows that DEX-treatment induces significant decrease in proliferation compared to Vehicle-treated tumors. B) Representative immunohistochemical staining for cleaved caspase-3 (an apoptotic marker) and H&E in vehicle- and DEX-treated mice. Corresponding bar graphs show quantification of % of CC3+positive cells and necrotic index (Necrotic index = Area of necrosis (μm^2)/Total area of tumor section (μm^2)* per 40X field within the tumor). No differences were observed with DEX treatment. N = 5 for all samples, p values were calculated with a Student's t-test. Scale bars: 50 μm for A.

SFigure 2. DEX does not suppress proliferation *in vitro.* (**A**) *In vitro* MTT assay of three independent primary murine glioma cultures treated with 0.1, 1 and 10 μM of DEX did not show a reduction in cell proliferation. (**B**) *In vitro* cell cycle analysis of primary murine glioma cultures treated with 3 days of 0.1, 1 and 10 μM of DEX showed that DEX induced a slight but significant increase in the G1 population (from 50.97% in vehicle to 54.19 - 54.82% with DEX), with a corresponding decrease in the G2 population (from 34.45% in vehicle to 30.17 - 30.53% with DEX). However, the magnitude of these changes was not as large as anticipated based on the *in vivo* BLI and IHC responses. p values were calculated using a one-way ANOVA in comparison to DMSO, * p<.05, **p<.01, *** p<0.001.

SFigure 3. B20-4.1.1 treatment has no effect on non-tumor vasculature, decreases edema by T1-contrast MRI and increases myeloid cell infiltration in contrast to DEX. A) Representative images of CD31 staining of a non-tumor cortical area in vehicle- and B20-4.1.1-treated mice and corresponding graphs showing no significant differences in either total vessel area or average vessel size between B20-4.1.1- and vehicle-treated mice. **B)** Representative images of T1-contrast MRI scans of vehicle- and B20-4.1.1-treated mice. Graphs comparing tumor volumes according to T1-contrast MRI scans in vehicle- and B20-4.1.1-treated mice at the endpoint of survival, demonstrating a statistically significant difference in tumor size between vehicle- and B20-4.1.1-treated animals. **C)** Representative images of Iba1 staining for tumor-

associated microglia/macrophages in vehicle and B20-4.1.1-treated mice (upper panel) and vehicle and DEX treated tumors. Graphs comparing the total Iba1 positive areas in vehicles and B20-4.1.1 and DEX treated mice show that while there is significant increase in macrophage infiltration in B20-4.1.1 treated mice compare to vehicle, no significant difference was observed in DEX-treated compare to vehicle-treated mice. p values were calculated using an unpaired Student's t-test, *p<0.05 **p < 0.01, ****p < 0.0001: Scale bars: 50 μm for A and C.

SFigure 4. Olig2-positive tumor cells showed a significant increase in cell death in response to B20-4.1.1 treatment. (A) Representative images of tumor sections and **(B)** corresponding quantification of cell death by TUNEL staining in Olig2-positive gliomas and CD31-positive endothelial cell populations. While there were no significant differences in the percentage of cell death of CD31-positive endothelial cells, there was a significant increase in the percentage of cell death of Olig2-positive tumor cells. n = 4 and 5 for vehicle and B20-4.1.1, respectively. p values were calculated with an unpaired Student's t-test, *p < 0.05. Scale bars: 50 μm.

STable 1. Patient and treatment characteristics, categorized by use of DEX at the start of radiotherapy. A) The median survival of all patients was 13.6 months (0.76-116.6 months) and 595 of the patients (96%) have died at the time of analysis. The median overall survival was 18.7, 18.4, 11.8, and 5.1 months in RTOG RPA classes III, IV, V, and VI, respectively (p<0.0001). **B)** Multivariate COX regression analysis of glioblastoma patients treated at MSKCC, examining RTOG RPA Class, concurrent TMZ, and DEX at the start of radiotherapy.

STable 2. Patient and treatment characteristics, categorized by use of DEX at the start of radiotherapy for EORTC (A) and GGN (B).

STable 3. A) PFS and OS in the GGN cohort by steroid use. B) Multivariate analysis of the association of steroid administration and outcome in the GGN cohort

STable 4. Genes and top predicted cellular functions and canonical pathways of DEX regulated gene set.. **A)** The 25 most significantly changed probes on an illumina mouse-ref-8 array. The gene list is based on a greater than 1.5 fold change, with an ANOVA p value <

0.0005. This list was generated using Partek Genomics Suite array analysis software (v6.6)**. B) Gene ontology analysis of DEX-regulated genes.** The top 5 most enriched predicted cellular function and canonical pathways, based on the 19 DEX-regulated genes.