Extended Data Fig. 5. Altered levels of tumor-specific antibody production and plasma cell differentiation occurring with both hIgG1-G396R and mIgG2c-G400R variant

(A) Secretion of IgG subclasses by activated TDLN B cells in stimulation with cell medium, irradiated LLC cells or MC38 cells. (B) TAA microarray analyses of IgG1 and IgG2b in the sera from untreated mouse (n=1), CAC-induced WT mice (n=5) and mIgG2c-G400R mice (n=5). (C) TAA microarray analyses of IgG3 in the plasma samples of healthy donor (n=1), WT CRC patients (n=6) and hIgG1-G396R homozygous CRC patients (n=6). (D) The ratios of IgG1/IgG3 were evaluated based on the TAA microarray results. (E) TAA microarray analyses of IgA and IgG2c subclasses in the colon explants from WT CAC mice (n=6), mIgG2c-G400R CAC mice (n=6) and cell medium. (F) OVA expression in MC38-mOVA cells and B16-mOVA cells detected by flow cytometry. The growth curves of B16-mOVA tumor cells in untreated mice and OVA-immunized mice are shown. (G) The MC38-mOVA tumor size in mIgG2c-tailless, WT and mIgG2c-G400R mice. Tumor tissues were isolated and weighed after euthanasia. (H) Representative flow cytometry plots of OVA-specific IgG2c+ GC B cells, plasma cells and memory B cells. OVA-specific IgG2c+ GC B cells were gated from total B220+ GL-7+ GC B cells, OVA-specific IgG2c+ memory B cells were gated from total B220+ CD38+ IgD+ memory B cells, and OVA-specific IgG2c+ plasma cells were gated from total B220low CD138high plasma cells. One of three representative experiments is shown (A, F, G, H). Statistical significance was determined using two-way ANOVA (F) and an unpaired two-tailed t-test (G). Mean± SEM.