Table W1. Source of Antibodies.

Antibody	Catalog No.
Depletion	
Anti–LFA-1 (αLβ2)	553118 (BD Pharmingen)
Anti–CD49d (α4)	553154 (BD Pharmingen)
Rat IgG2a	553927 (BD Pharmingen)
Rat IgG2b	553986 (BD Pharmingen)
Flow cytometry	
Anti-CD45	560501 (BD Pharmingen)
Anti-CD19	561113 (BD Pharmingen)
Anti-CD138	553714 (BD Pharmingen)
Anti-CD40	558695 (BD Pharmingen)
Anti-CD86	553768 (BD Pharmingen)
Anti-CD21	552957 (BD Pharmingen)
Anti-B220	553138 (BD Pharmingen)
Anti-CD3	553063 (BD Pharmingen)
Anti-CD8 alpha	553035 (BD Pharmingen)
Anti-CD4	553052 (BD Pharmingen)
Anti-CD14	553063 (BD Pharmingen)
Immunocytochemistry	
Anti-CD19	MCA1439GA (Serotec, Oxford, United Kingdom)
Anti-vimentin	NB100-92123 (Novus Biological, Littleton, CO)
$Alexa_{594}$-conjugated goat anti-rat IgG	A-11007 (Invitrogen)
$Alexa_{488}$-conjugated goat anti-rabbit IgG	A-11034 (Invitrogen)

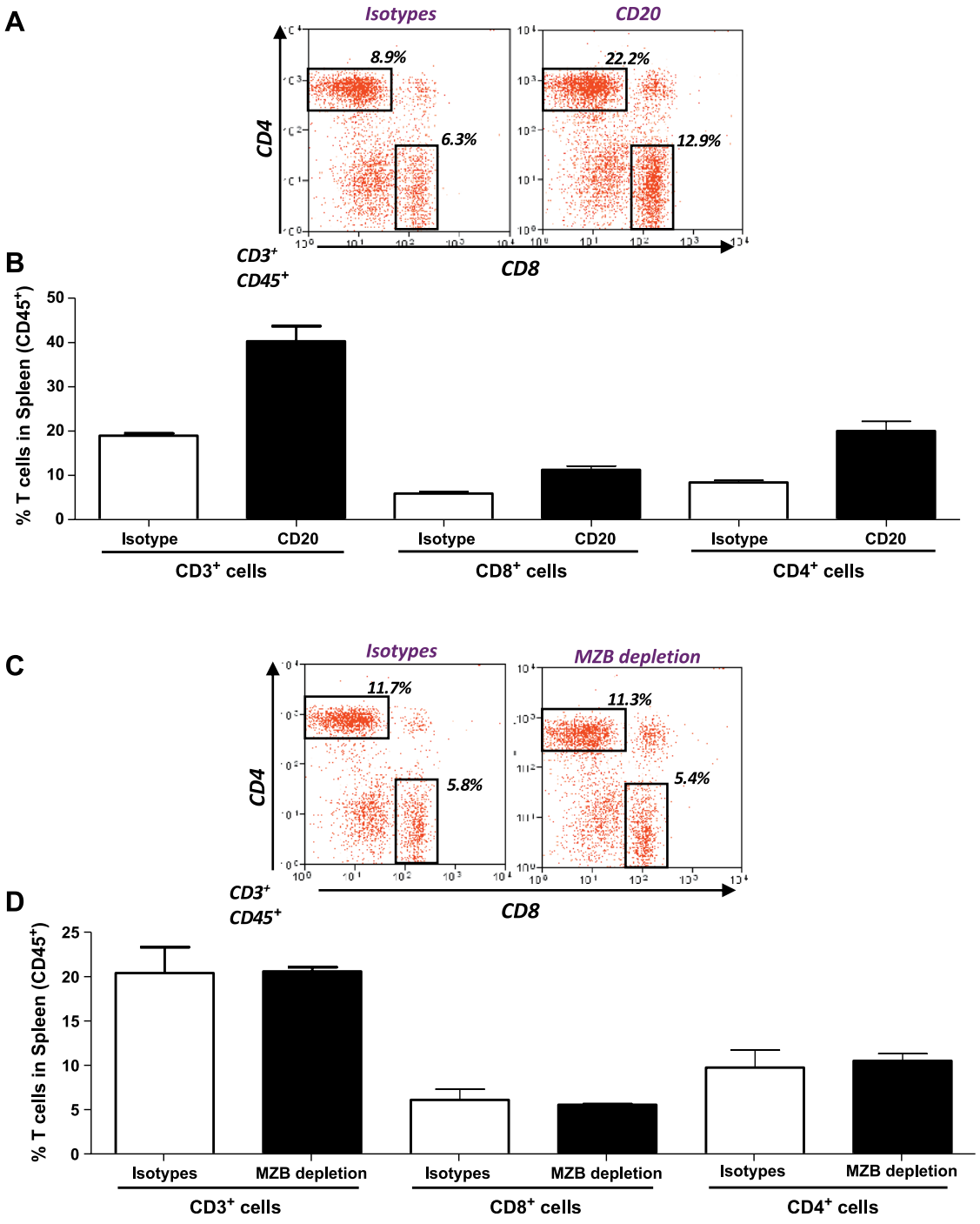

Figure W1. Administration of antimouse CD20 antibodies or MZB depleting antibodies does not deplete T cells. (A) Representative dot plots show the populations of T lymphocytes in the spleen of naive mice injected i.p. with mouse antimouse CD20 antibody (IgG2a, 300 μg) or with isotype control (mouse anti-human CD20 IgG2a, 300 μg). (B) Spleens were collected 7 days after depletion and total $CD3^+$ lymphocytes, $CD4^+$ T cells, and $CD8^+$ T cells ($CD45^+/CD3^+$ and $CD4^+$ or $CD8^+$, respectively) were quantified by flow cytometry. (C) Representative dot plots show the populations of T lymphocytes in the spleen of naive mice injected i.p. with anti-CD49d antibody (100 μg) and anti–LFA-1 antibody (100 μg) or the corresponding isotype controls (rat IgG2a and rat IgG2b, 100 μg each). (D) Spleens were collected 7 days after depletion, and total $CD3^+$ lymphocytes and CD4 and CD8 T cells ($CD45^+/CD3^+$ and $CD4^+$ or $CD8^+$), respectively, were quantified by flow cytometry.

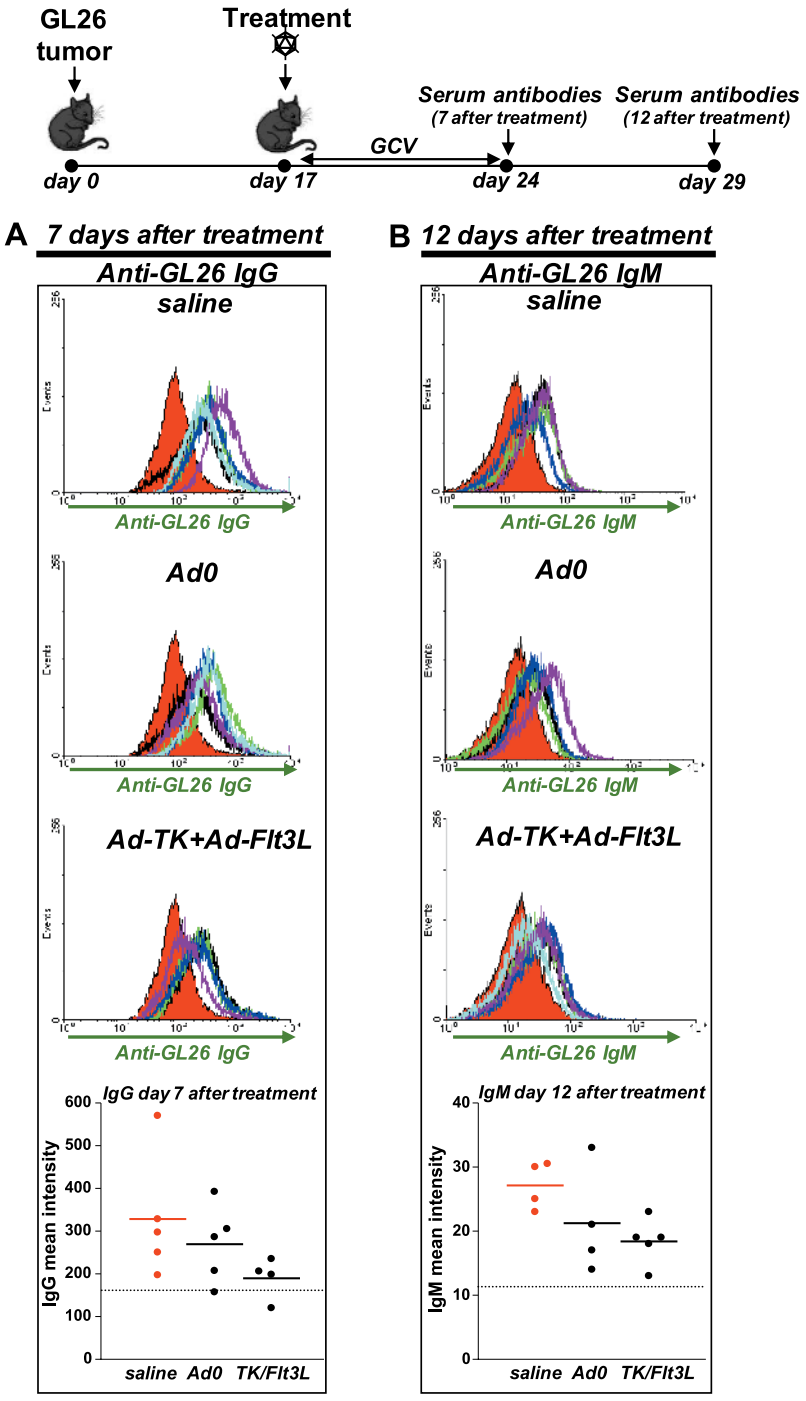

Figure W2. Assessment of the antibody response against the tumor. GL26 cells were implanted in the striatum of C57BL/6 mice and treated 14 days later with an intratumoral injection of Ad-TK+Ad-Flt3L, saline, or an empty vector (Ad.0). At 7 (A) and 12 (B) days after the treatment, serum was collected to evaluate the presence of circulating anti-GL26 cell IgG (A) and IgM (B), respectively. Histograms and graphs show the fluorescence intensity of fixed GL26 cells that were incubated with normal C57BL/6 serum (red area) or serum from treated tumor-bearing mice (colored lines), followed by FITC-conjugated antimouse IgM or IgG.

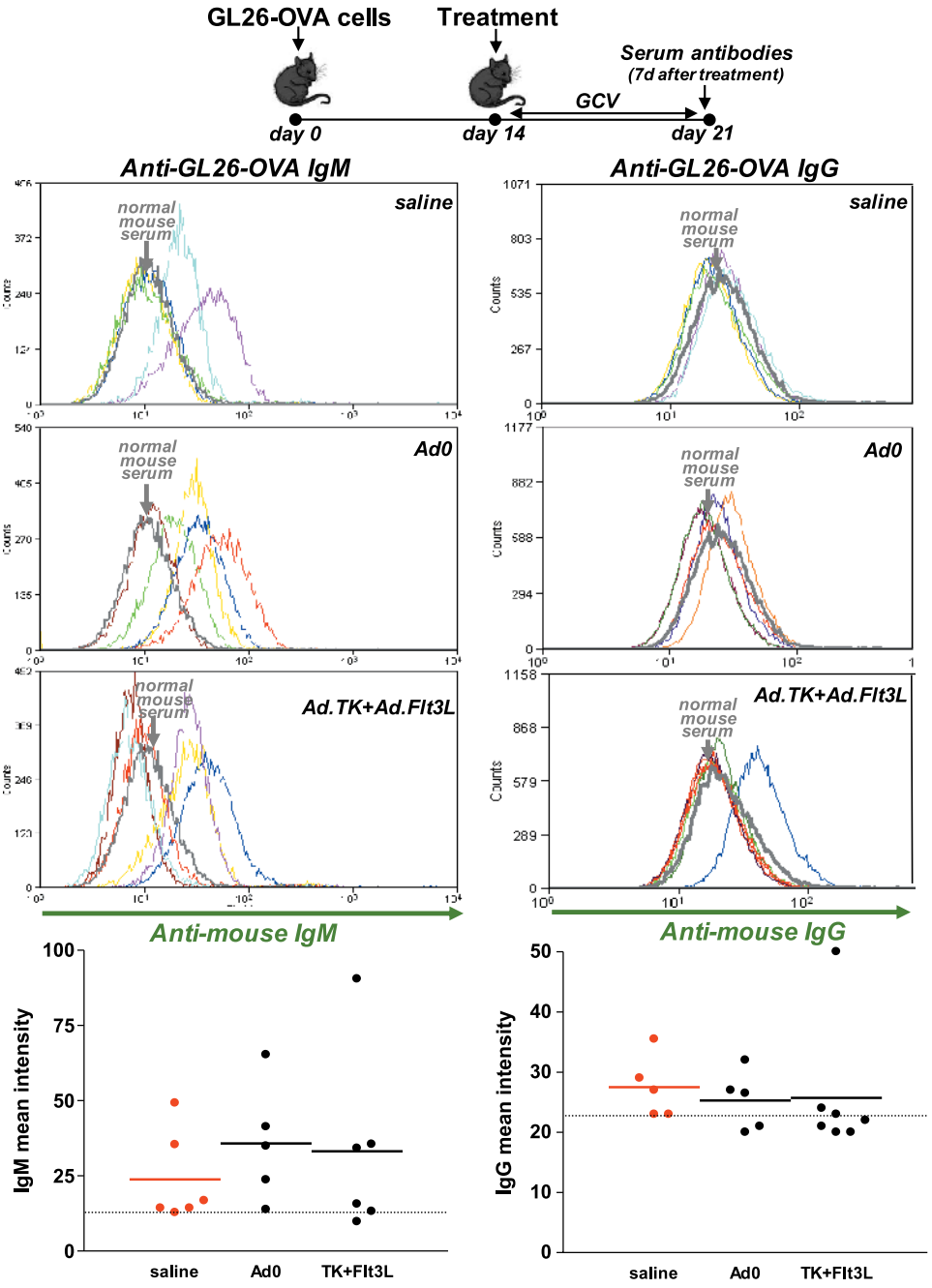

Figure W3. Assessment of the antibody response against tumor cells expressing a surrogate antigen. GL26 cells expressing chicken ovalbumin (GL26-OVA) were implanted in the striatum of C57BL/6 mice and treated 14 days later with an intratumoral injection of Ad-TK+Ad-Flt3L, saline, or an empty vector (Ad.0). Seven days after the treatment, serum was collected to evaluate the presence of circulating anti–GL26-OVA cell IgM and IgG. Histograms and graphs show the fluorescence intensity of fixed GL26-OVA cells that were incubated with normal C57BL/6 serum (red area) or serum from treated tumor-bearing mice (colored lines), followed by FITC-conjugated antimouse IgM or IgG.

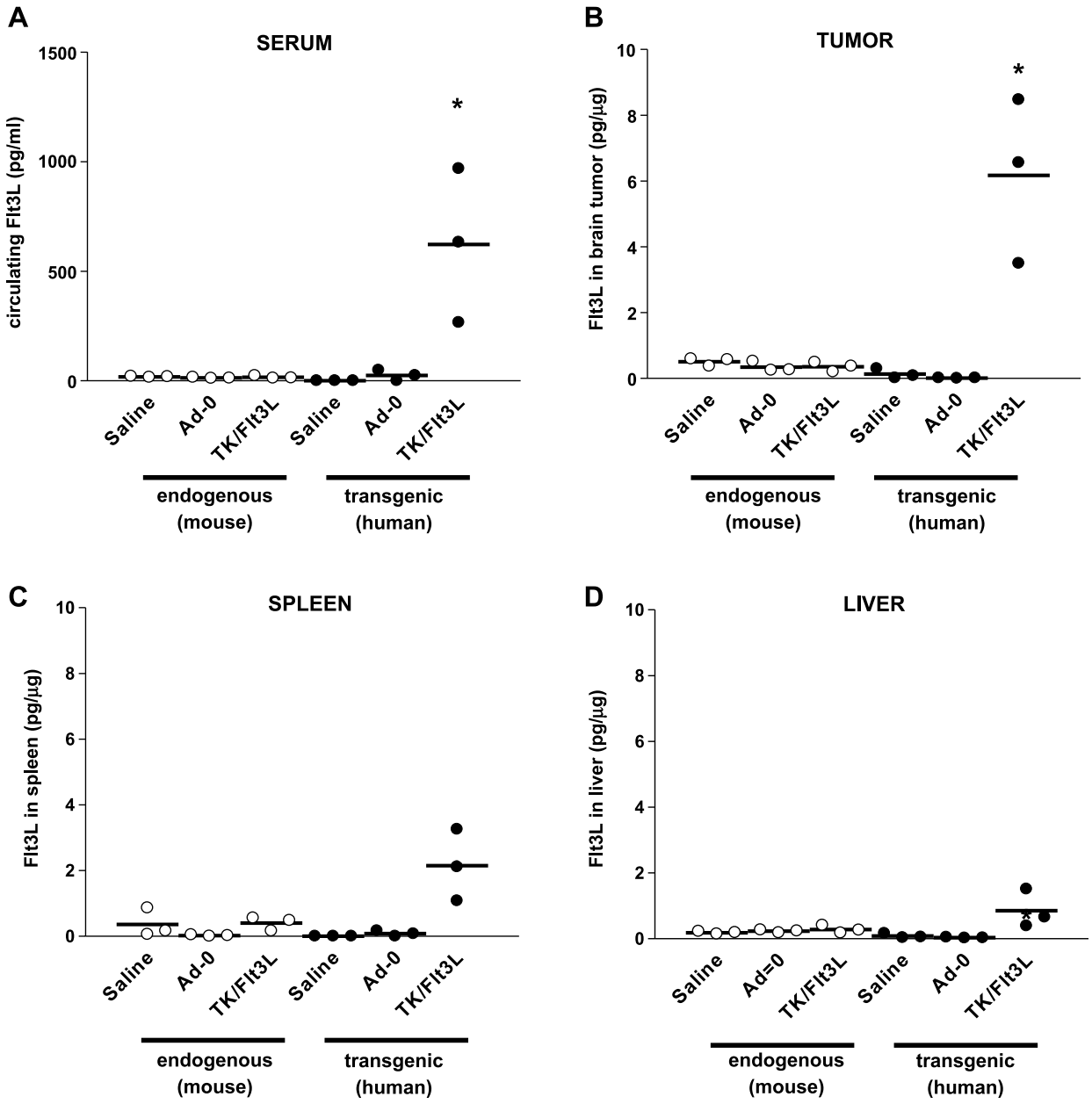

Figure W4. Flt3L expression 7 days after intratumoral delivery of Ad-Flt3L and Ad-TK. (A) GL26 tumor cells were implanted into the striatum of C57BL/6 mice; tumors were treated 14 days later with saline (S), Ad.0, or Ad-TK+Ad-Flt3L (TF). Levels of Flt3L expressed from the Ad vector were assessed using an ELISA specific for human Flt3L (transgenic). Levels of endogenous Flt3L were assessed using an ELISA specific for mouse Flt3L. Serum (A), brain tumors (B), spleen (C), and liver (D) were harvested 7 days after treatment, and both endogenous and transgenic Flt3L levels were assayed by ELISA. *$P < .05$ *versus* corresponding saline. Two-way ANOVA followed by the Tukey test.