Fig.S1

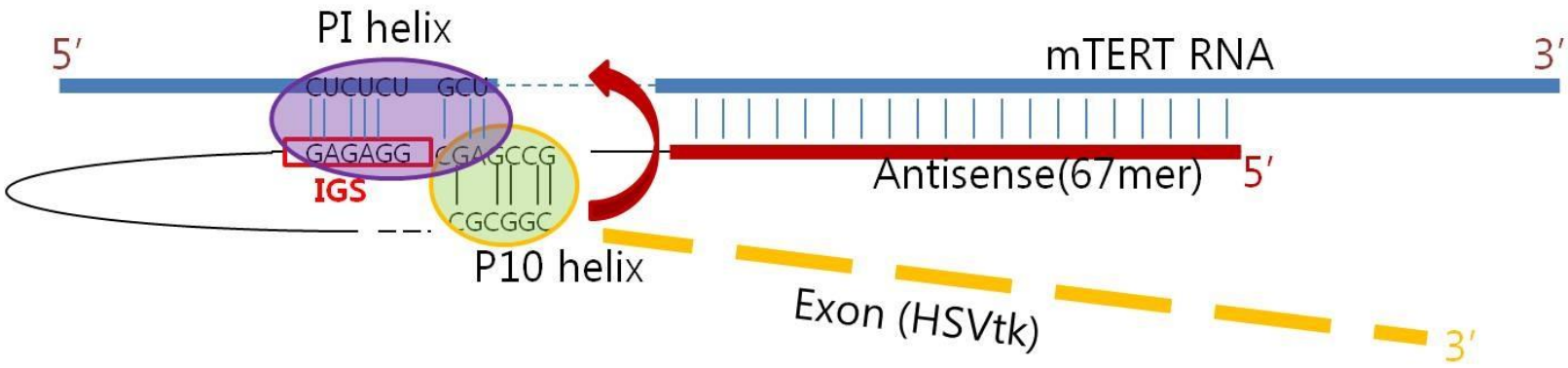

Fig.S1. Schematic diagram of the mouse TERT-targeting *trans*-splicing ribozyme (mTERT-TR)-HSVtk construct. The target site on the mouse TERT(mTERT) transcript is represented by sequences around the splicing site (blue and red lines). The *trans*-splicing ribozyme recognizes mTERT RNA specifically using anti-sense mTERT RNA sequence (red line; 67 mer) and digests mTERT RNA at 3' end of the internal-guided-sequence (IGS). Then 5' end of HSVtk coding RNA part of the *trans*-splicing ribozyme (yellow line) is ligated to the digested end of mTERT RNA (indicated as red arrow). Potential base pa irings between the mTERT target mRNA and ribozyme are indicated by vertical lines.

Fig.S2

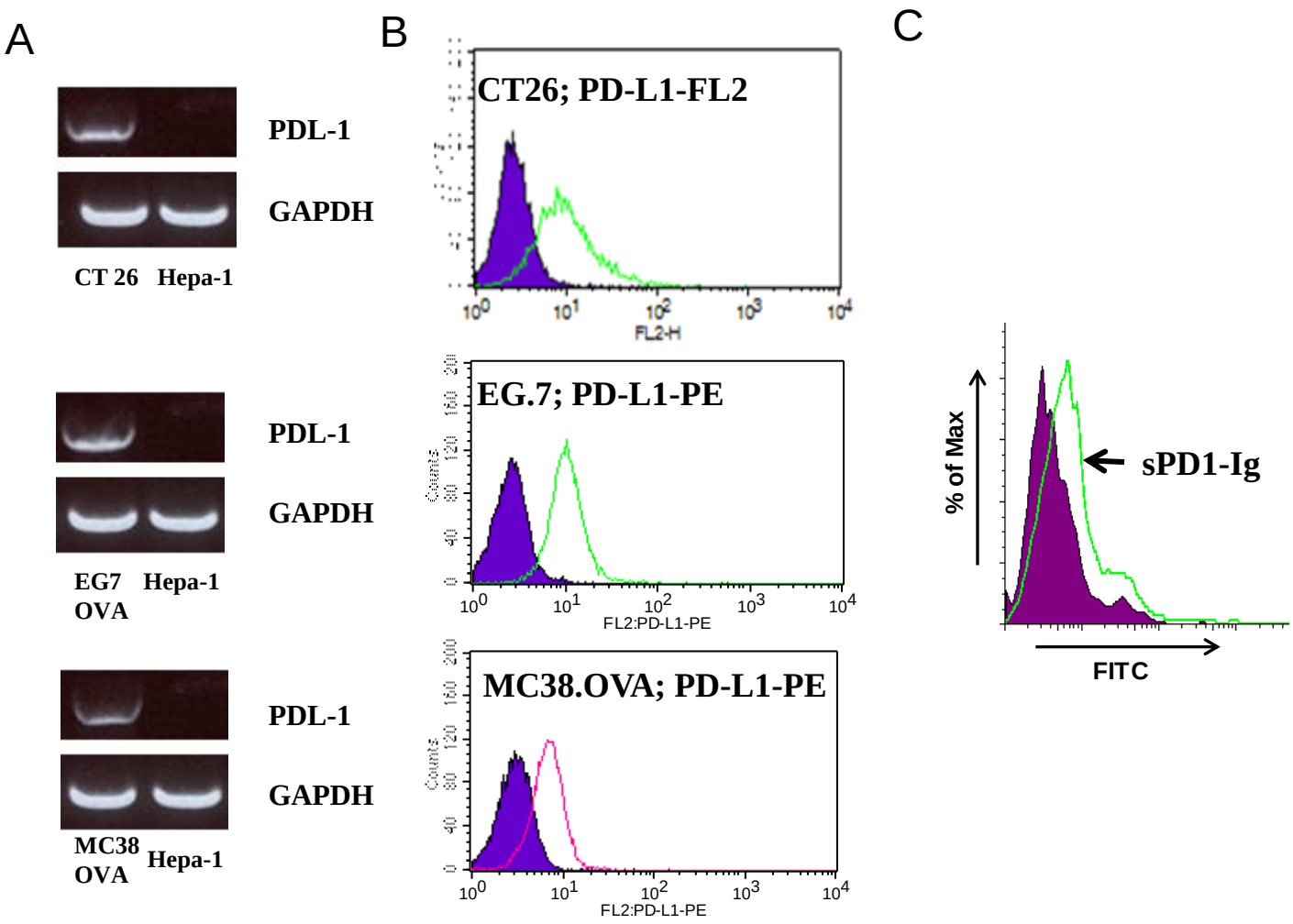

Fig.S2. PD-L1 expression on the surface of mouse cancer cells. a. Total RNA was prepared from each cell and PD-L1 transc ripts were detected by RT-PCR. Mouse liver cancer cells (Hepa-1) were analyzed in parallel for comparison. **b.** PD-L1 proteins o n the surface of each cell was analyzed by flow cytometry. **c.** The culture supernatant from Ad5mTR.sPD1-infected 293 cells was incubated with E.G7 cells and then sPD1-Ig proteins on the cell membrane were detected with anti-mouse FITC-conjugated antibody. The cells either treated with the secondary antibody alone (filled area) or with the culture supernatant along with the secondary antibody (open area) were analyzed by flow cytometry.

Fig.S3

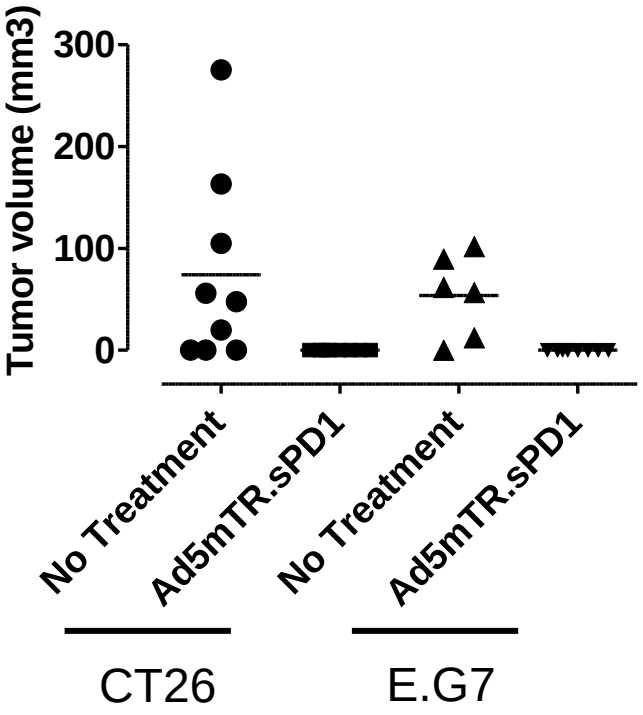

Fig.S3. Inhibition of secondary tumors by Ad5mTR.sPD1 treatment. When the primary subcutaneous CT26 tumors in BALB/c mice were palpable (the average tumor size of 57.53 mm^3), the mice were injected intratumorally with 5 × 10^8 PFU of Ad5mTR.sPD1 three times at 3-day intervals. Two days after the final treatment, the opposite flank was challenged with 1 × 10^6 of CT26 cells. After 14 days, the individual tumor size at the second site was plotted above (Ad5mTR.sPD1). For the control group, when the primary CT26 tumors became palpable (the average size of 97.88 mm^3) without adenoviral injection, the opposite flank was challenged with 1 × 10^6 of CT26 cells. After 14 days, the individual tumor size at the second site was plotted above (no treatment). In case of E.G7, when the primary subcutaneous E.G7 tumors implanted in C57/BL6 mice were palpable (average tumor size of 41.40 mm^3), the mice were injected intratumorally with Ad5mTR.sPD1 as described for CT26 tumors. Two days after the final treatment, the opposite flank was challenged with 1 × 10^6 of E.G7 cells. After 10 days, the second tumor growth was completely suppressed as shown above (Ad5mTR.sPD1). For the control group, when the primary E.G7 tumors implanted subcutaneously in C57/BL6 mice were palpable (the average tumor size, 54.55 mm^3) without adenoviral injection, the opposite flank was challenged with 1 × 10^6 of E.G7 cells. After 10 days, the individual tumor size at the second site was plotted above (no treatment).