Table S1. Immunogenic CT26 mutation percentage

Gene	Mutated sequence	Substitution (WT, AA#, Mut)	Reactive T cell subtype	Mutation percentage
Slc20a1	DKPLRRNNSYTSYIMAICGMPLDSFRA	T425I	CD4	50%▲
Nphp3	AGTQCEYWASRALDSEHSIGSMIQLPQ	G234D	CD4	50%▼
Slc4a3	PLLPFYPPDEALEIGLELNSSALPPTE	T373I	CD4	50%▲
Cxcr7	MKAFIFKYSAKTGFTKLIDASRVSETE	L340F	CD4	100%
E2f8	VILPQAPSGPSYATYLQPAQAQMLTPP	I522T	CD8	50%▼
Agxt2l2	EHIHRAGGLFVADAIQVGFGRIGKHFW	E247A	CD4	50%▼
Nap1l4	No mutation	-	CD4	-
Dhx35	EVIQTSKYYMRDVIAIESAWLLELAPH	T646I	CD4	50%
Als2	No mutation	-	CD4	-
Deptor	SHDSRKSTSFMSVNPSKEIKIVSAVRR	S253N	CD4	100%
Tdg	AAYKGHHYPGPGNYFWKCLFMSGLSEV	H169Y	CD4	50%▲
Rpap2	CGYPLCQKKLGVISKQKYRISTKTNKV	P113S	CD4	50%▼
Steap2	VTSIPSVSNALNWKEFSFIQSTLGYVA	R388K	CD4	50%
Nbea	PAPRAVLTGHDHEIVCVSVCAELGLVI	V576I	CD4	100%
Aldh18a1	LHSGQNHLKEMAISVLEARACAAAGQS	P154S	CD4	50%▲
Zc3h14	NCKYDTKCTKADCLFTHMSRRASILTP	P497L	CD4	50%▼
Drosha	LRSSLVNNRTQAKIAEELGMQEYAITN	V1189I	CD4	100%

The mutation percentage of genes was confirmed based on the study of mutant neoantigens that induce a therapeutic immune response to cancer in another group [31].
Bold text is mutated sequence site. Symbol ▲; more than, symbol ▼; below

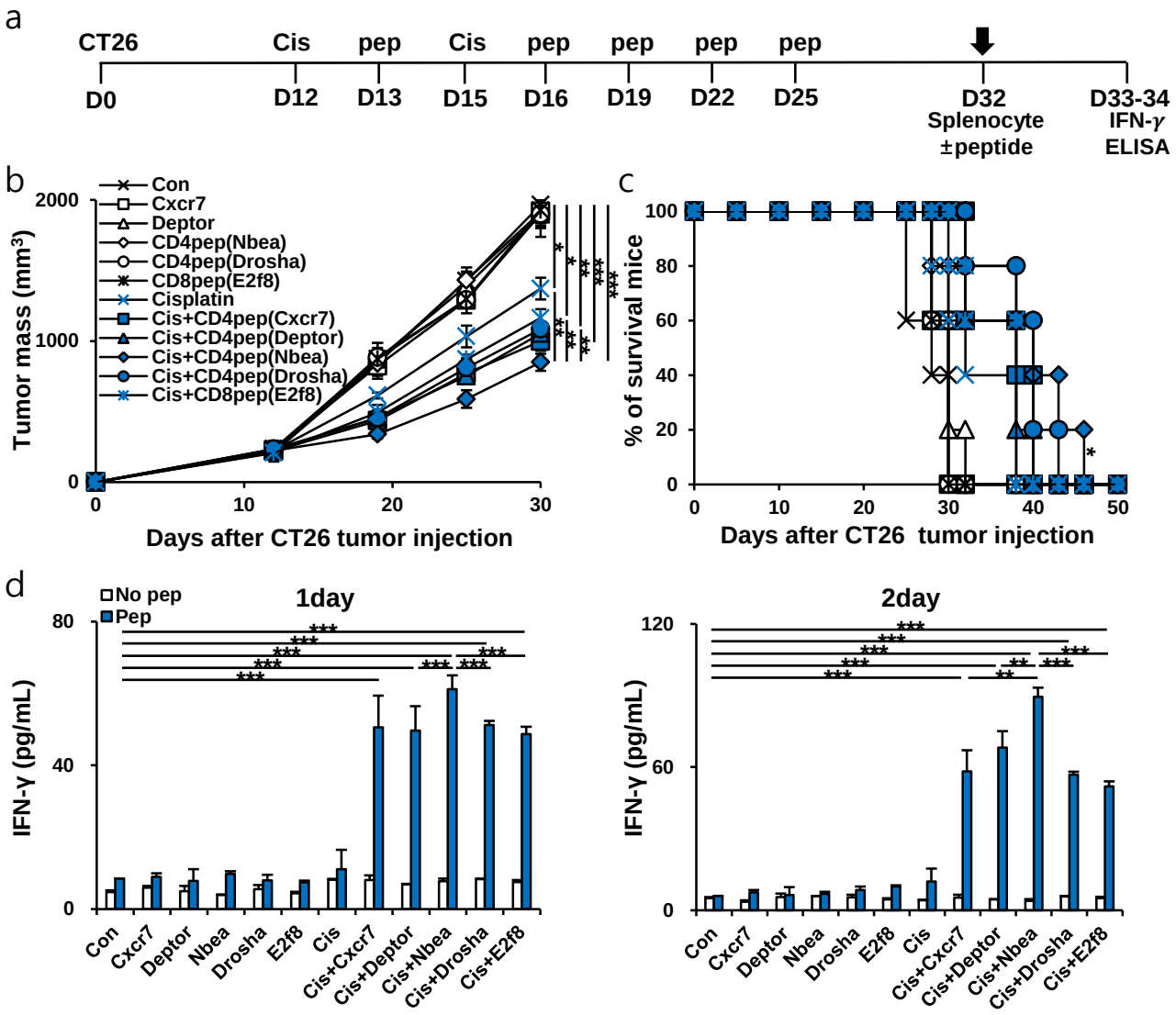

Figure S1. Tumor treatment effects of neoantigen vaccine in cisplatin-treated mouse model.
In the *in vivo* experiments, BALB/c mice were injected with 2 × 10^5 CT26 cells (per mouse) subcutaneously and were treated intraperitoneally with 5 mg/kg cisplatin on days 12 and 15. Then, the mice were treated intratumorally with 20 μg (per mouse) of $CD4^+$ or $CD8^+$ T cell neoantigen peptide on days 13, 16, 19, 22, and 25. (a) Schedule flowchart (b) Tumor mass was measured until the mice died or the diameter was over 2 cm (n = 5). (c) Survival of the mice was observed for 50 days (n = 5). (d) After a week of the last vaccination, splenocytes were isolated and re-stimulated with $CD4^+$ or $CD8^+$ T cell neoantigen peptide for 1 or 2 days, and the levels of IFN-γ were estimated by ELISA. IBM SPSS Statistics Base 22.0 was used as the statistical tool to analyze the differences between the groups. *P<0.05, **P<0.01, ***P<0.001.

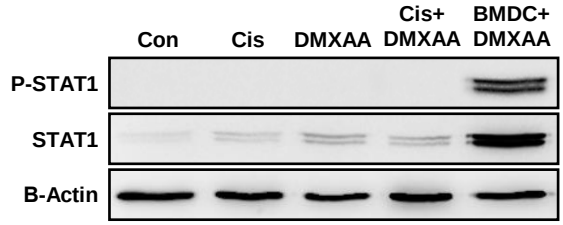

Figure S2. Phosphorylation and activation of STAT1 induced by STING and type 1 IFN signaling.
TC-1 tumor cells were treated with/without cisplatin (20 μg/ml) for 2-3h, wash out, and/or DMXAA (10 μg/ml) or DMXAA-treated C57BL/6 mouse BMDCs supernatants were incubated overnight. Cells were harvested, lysed, and then evaluated for expression of STAT1 using Western blot analysis.

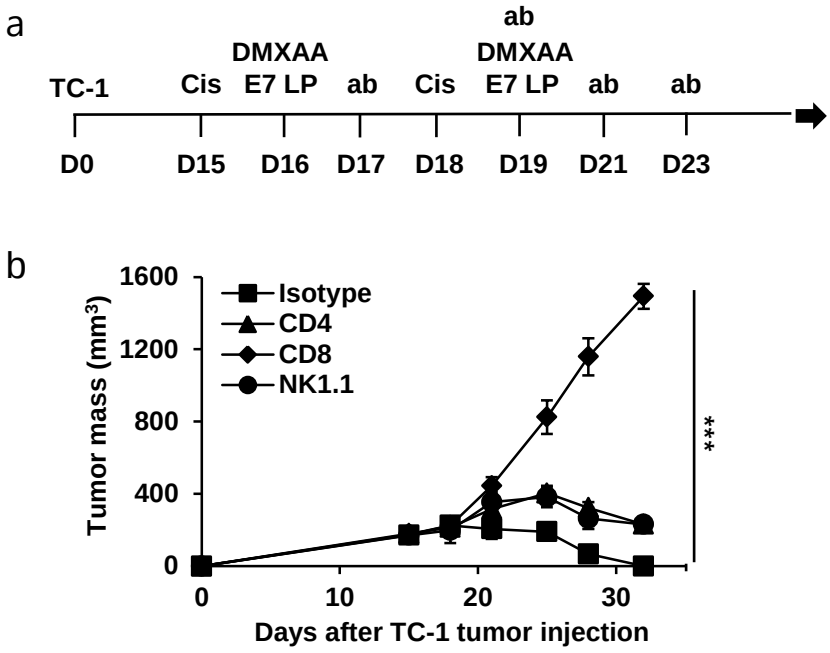

Figure S3. Direct tumor killing effect of CD8 T cells.
C57BL/6 mice were injected subcutaneously with 2 × 10^5 TC-1 cells/mice on day 0. Mice were then treated and/or intraperitoneally with 5 mg/kg cisplatin on days 15 and 18, intratumorally with 20 μg/mice of E7 long peptide with 100 μg/mice of DMXAA on days 16 and 19, 100μg/mice of control isotype IgG or anti-CD4 or anti-CD8 or anti-NK depletion antibodies were injected intraperitoneally on days 17, 19, 21 and 23. (a) schedule flowchart (b)Tumor mass was measured until the mice died or the diameter was over 2 cm (n = 5). IBM SPSS Statistics Base 22.0 was used. *P<0.05, **P<0.01, ***P<0.001.

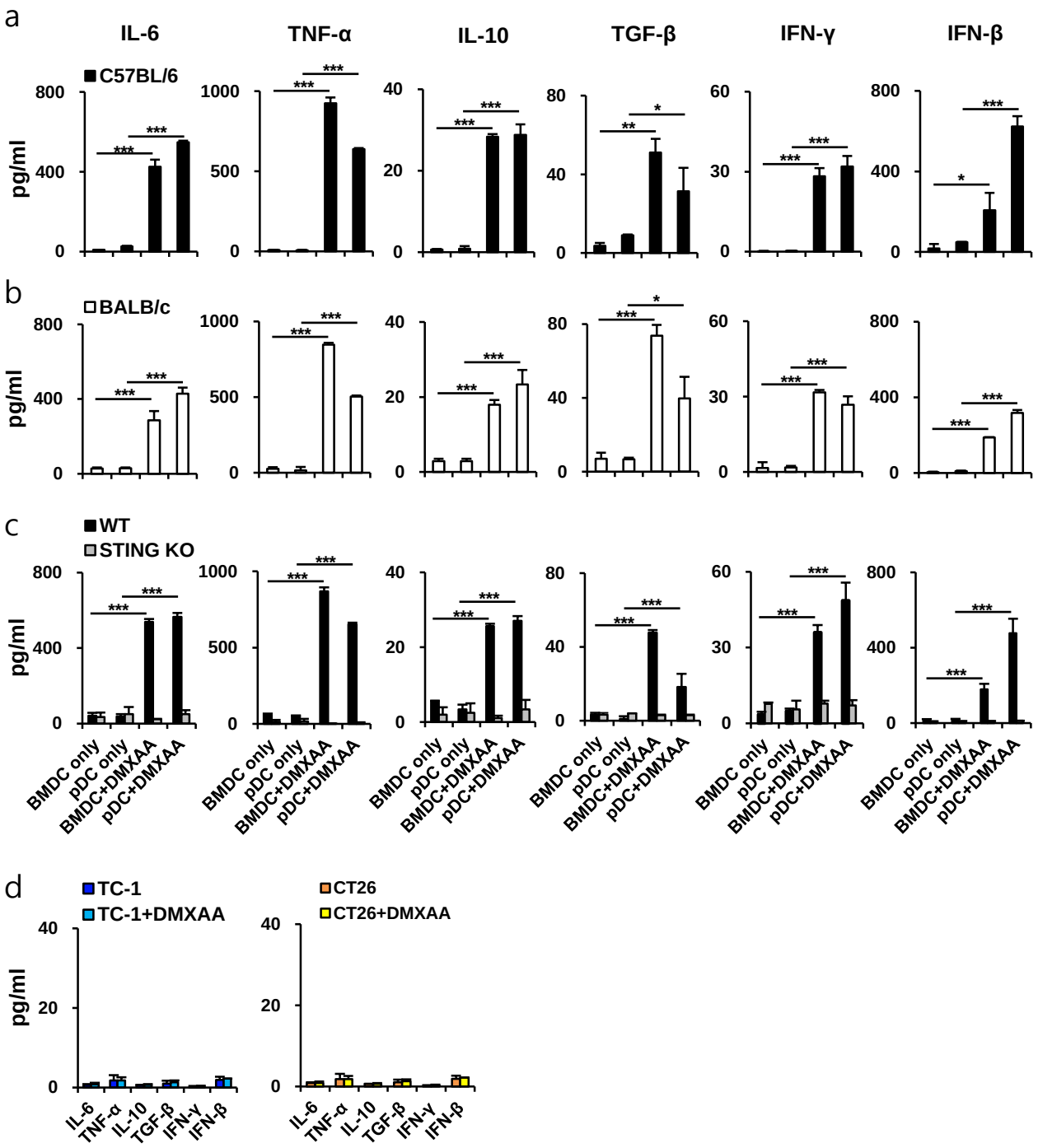

Figure S4. Variose cytokines increases directly due to the presence and activation of STING and APC.
BMDC and pDC of C57BL/6, BALB/c and STING KO mice, TC-1 and CT26 tumor cells were treated with 5 μg/ml of DMXAA. After incubation for 24 h, the supernatants were harvested and assessed for cytokines levels using a mouse IL-6, TNF-α, IL-10, TGF-β, IFN-γ and IFN-β ELISA. (a) ELISA cytokine levels graph performed on BMDC and pDC of C57BL/6 mice (n = 3). (b) ELISA cytokine levels graph performed on BMDC and pDC of BALB/c mice (n = 3). (c) ELISA cytokine levels graph performed on BMDC and pDC of C57BL/6 (WT) and STING Knock out mice (n = 3). (d) ELISA cytokine levels graph performed on TC-1 and CT26 tumor cells (n = 3). IBM SPSS Statistics Base 22.0 was used. *P<0.05, **P<0.01, ***P<0.001.

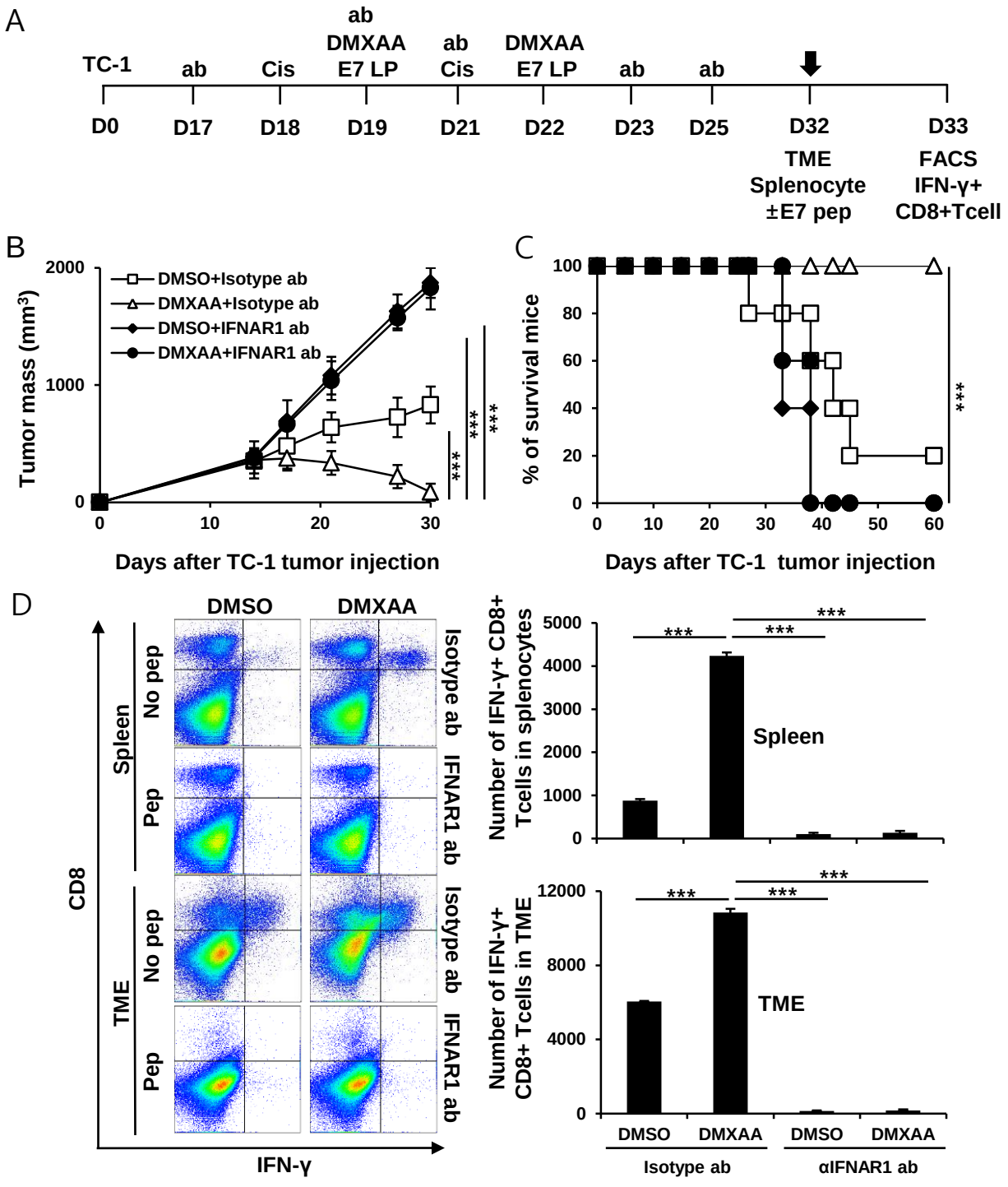

Figure S5. Blocking of type Ⅰ IFN reduces the anticancer immune response.
C57BL/6 mice were injected with 2 × 10^5 TC-1 cells/mice subcutaneously on day 0. Mice were then treated intraperitoneally with 5 mg/kg cisplatin on days 18 and 21, intratumorally with 20 μg/mice of E7 long peptide and/or with 100 μg/mice of DMXAA on days 19 and 22. Also, treated intraperitoneally with 100 μg/mice of isotype or IFNAR1 antibody on days 17, 19, 21, 23 and 25. (A) Schedule flowchart. (B) Tumor mass was measured until the mice died or the diameter was over 2 cm (n = 5). (C) Survival of the mice was observed for 60 days (n = 5). (D) After a week of the last vaccination, tumor tissues and spleens of TC-1 tumor-bearing mice were harvested and re-stimulated with E7 short peptide, and then analyzed of IFN-γ^+ $CD8^+$ T cells by flow cytometry (n = 3). IBM SPSS Statistics Base 22.0 was used. *P<0.05, **P<0.01, ***P<0.001.