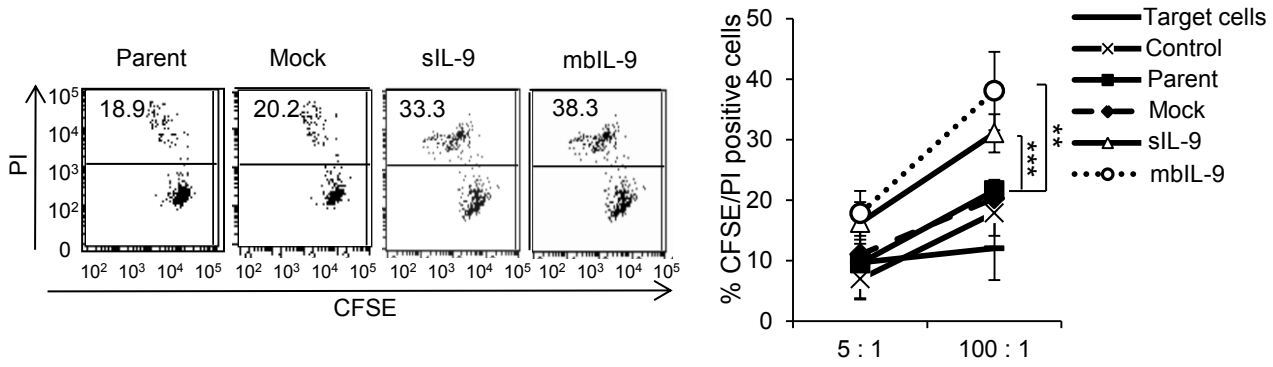
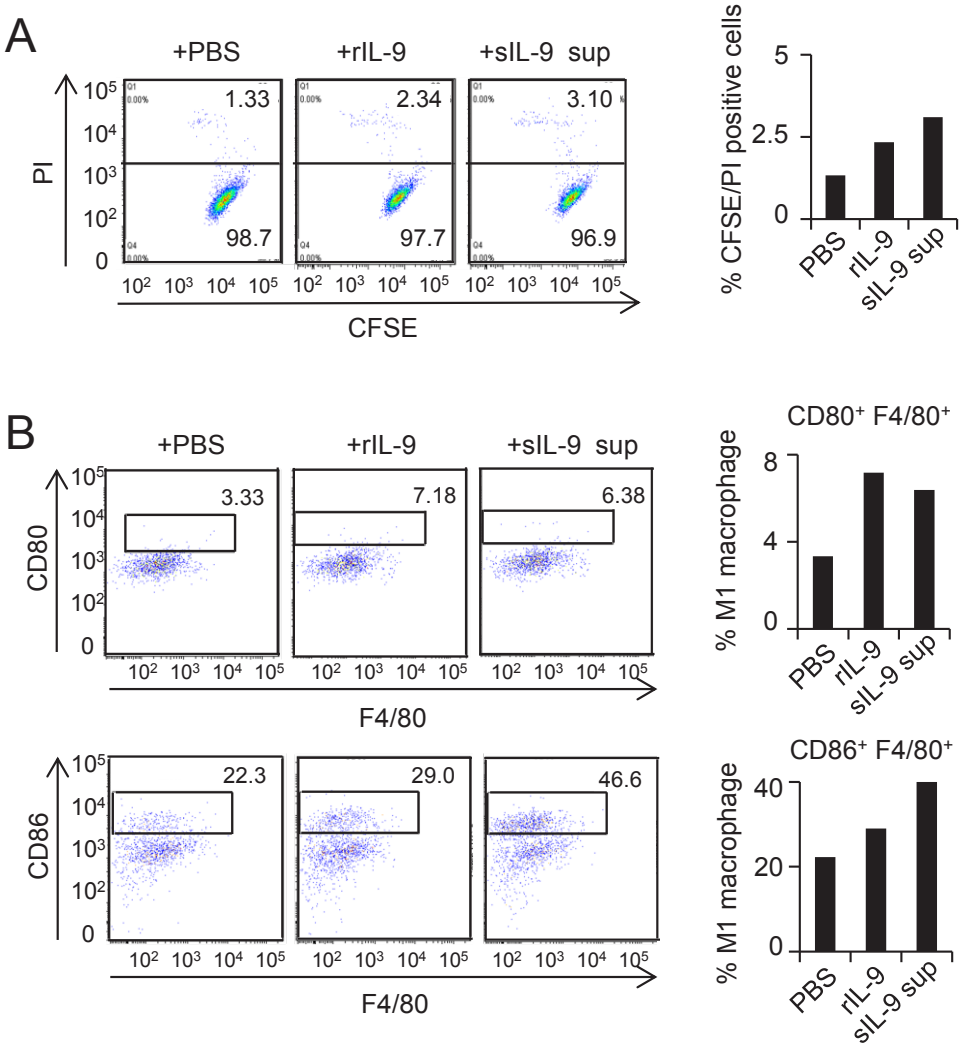
Supplementary Table S1. Primers used in this study

TNF-α	F	5’ GCG GTA CCA TGA GCA CAG AAA GCA TG 3’
	R	5’ CGC GAA TTC CTC CGG CCA TAG AAC TGA T 3’
sIL-9	F	5’-CAG GAT CCC GTC AAC ATG TTG GT-3’
	R	5’-GCG CTC GAG TCA TGG TCG GCT TTT CTG CCT-3’
mbIL-9	F	5’-CG CGA ATT CAG AGA TGC AGC ACC ACA TGG-3’
IL-9Rα	F	5'-GGA CAG GAA CAG GTC AGC CTG GTG-3'
	R	5'-GTA CAC ACT GTA GAG AGG ATG GAA GAA-3'

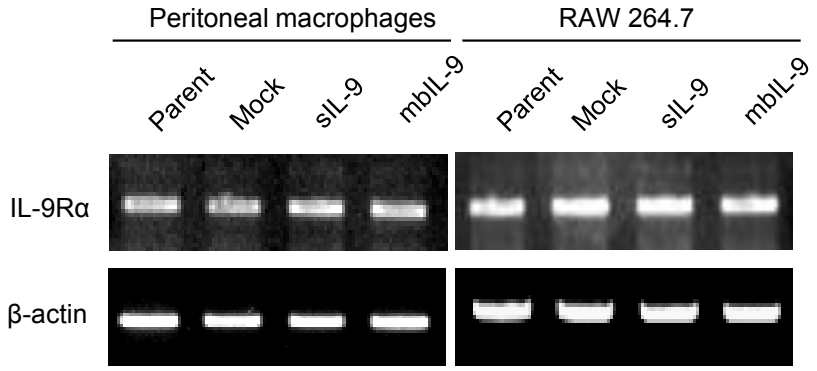
F : Forward, R : Reverse



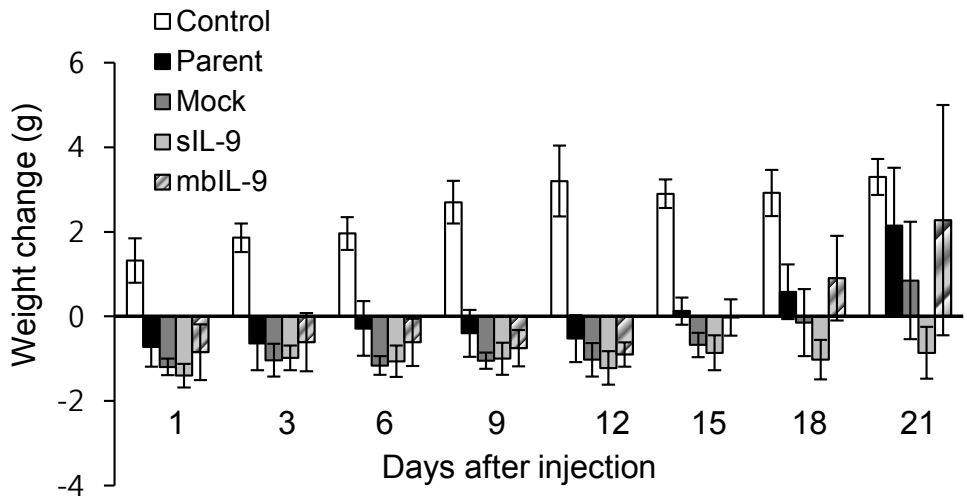
Supplementary Fig. S1. IL-9 exerted its anti-tumor effect through stimulating cytotoxic activity of splenocytes. Pre-immunized spleen cells were stimulated with MMC-inactivated IL-9 expressing B16F10 cells (ratio 5:1) for 48 h. The stimulated splenocytes were mix-cultured with CFSE-labelled wild-type B16F10 cells at ratio of 5:1 or 100:1 (left panel). After 4 h, the cell mixtures were harvested and stained with PI. Target cells; CFSE-labelled wild-type B16F10 cells only. Control; Non-stimulated splenocytes were mix-cultured with CFSE-labelled B16F10.



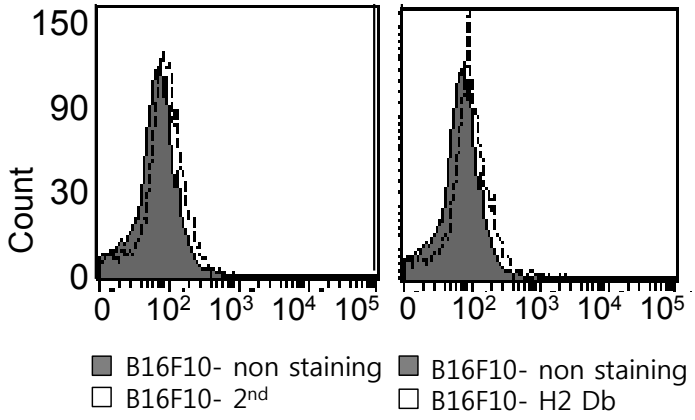
Supplementary Fig. S2. Recombinant IL-9 stimulated cytotoxic activity of macrophages. (A) RAW264.7 macrophages were stimulated with either PBS only, recombinant IL-9 (rIL-9, 0.3 ng/ml) or culture supernatants from sIL-9 clones for 24 h. The stimulated RAW 264.7 were mix-cultured with CFSE-labelled wild-type B16F10 cells at ratio 4:1. After 4 h, the cell mixtures were harvested and stained with PI. The percentage of dead cells was presented as a bar graph. (B) RAW264.7 macrophages were stimulated as (A) for 24 h and analyzed by FACS staining with the indicated antibodies. The percentage of M1 macrophages were presented as bar graphs.



Supplementary Fig. S3. IL-9 receptor expression on peritoneal macrophages and RAW264.7 cells. Peritoneal and RAW264.7 macrophages were treated with supernatants of mock control, sIL-9, and mIL-9 clones for 24 h. Then, the mRNA expression of IL-9Rα in the non-treated and treated cells were analyzed by RT-PCR.



Supplementary Fig. S4. Bodyweight change of mice injected with transfected B16F10 clones. C57BL/6 mice (n = 5) were subcutaneously implanted with tumor clones (5×10^4 cells/mouse). A group of same age mice (n = 5) without tumor implantation was used as a control for body weight change. The body weight was monitored daily and shown in the graph.



Supplementary Fig. S5. Surface MHC class I expression on B16F10 melanoma cells. MHC class I was stained with primary anti-MHC class I (H2 Db) antibody.