Supplementary figure legends

Supplementary figure 1A. Binding of mch1N11 antibody to PS in the presence of mouse beta 2-glycoprotein I. PS was adsorbed to 96-well plates. After blocking with 10% BSA, mch1N11 or control antibodies were added in the presence or absence of human or mouse beta 2-glycoprotein I. The plates were washed, and the bound antibody was detected using appropriate secondary antibodies labelled with HRP. Plates were developed by chromogenic substrate O-phenylenediamine followed by reading plates at 490 nm using a microplate reader.

Supplementary figure 1B. Binding of mch1N11 antibody to PS but not to phosphatidylcholine (PC), phosphatidylethanolamine (PE) and sphingomyelin (SM). Phospholipids were adsorbed to 96-well plates. ELISA was performed as described above in A).

Supplementary figure 1C. Binding of mch1N11 antibody to PS in the presence of human or mouse beta 2-glycoprotein I by flow cytometry. PS positive apoptotic B16 cells was induced by irradiation (100 Gray). Cells were pre-incubated with anti-CD16/32 (clone 2.4G2) at 4ºC for 30 min prior to antibody staining. mch1N11 were added in the presence or absence of human or mouse beta 2-glycoprotein I. The bound antibody was detected using FITC-labeled goat anti-mouse antibody. Cells were then analyzed using a FACSCalibur flow cytometer.

Supplementary figure 1D. Multilamellar vesicles (1 mg/mL of 50 % PS in PC) were incubated for 30 min at 20ºC with mch1N11 (20 μg/ml) in the presence or absence of beta 2-glycoprotein I (7 μg/ml). The tubes were vortexed and precipitates of immune complex were monitored. An aliquot was mounted on a microscope slide and photographed at 100X.

Supplementary Figure 2. Quantitative immunohistochemistry of K1735 tumors following combination therapy of PS-targeting antibody and anti-PD-1 antibody. Frozen sections were stained for CD8, F4/80 and Arg-1. Slides were developed with DAB substrate and counterstained using hematoxylin. Slides were digitally scanned to quantitate the specific staining. Graphs represent the intensity of peroxidase staining of representative sections from individual tumors along with mean and standard error. Statistical significance was determined by t-test using Graph Pad.

Supplementary figure 3. Enhanced Production of IFNγ by Splenocytes of Combination Therapy-treated Animals is Dependent on Presence of Tumor. Splenocytes prepared from B16 tumor-bearing mice (N=5) treated on days 3, 7, and 10 with spleens harvested 12 days after tumor implantation. Splenocytes from a matching set naïve animals (N=5) injected with the same antibody dose and frequency as tumor bearing animals were prepared 2 days after the last injection. IFNγ ELISpot was performed as described in methods. Data is presented as mean ± sem. Significant differences between treatment groups were determined by ANOVA.

Supplementary Table

Table 1. Antibodies for flow cytometry and immunohistochemistry

Target Molecule	Antibody Clone	Vendor
CD3	145-2C11	eBioscience
CD4	GK1.5	eBioscience
CD8a	53-6.7	eBioscience
CD11b	M1/70	eBioscience
CD11c	N418	eBioscience
CD45	30-F11	eBioscience
CD137	17B5	eBioscience
F4/80	BM8	eBioscience
FoxP3	FJK-16s	eBioscience
Gr-1	RB6-8C5	eBioscience
Granzyme B	NGZB	eBioscience
IFNγ	XMG1.2	eBioscience
Ki-67	SolA15	eBioscience
TNFα	MP6-XT22	eBioscience
NOS2	CXNFT	eBioscience
Arg-1	Sheep anti-mouse	R&D systems