**Supplementary Table and Figure Legends**

**Supplementary Table 1**

Pathway analyses identified the top 100 upregulated pathways related to immune response following administration of chemotherapy (paclitaxel, PTX, n=20; carboplatin, CBDCA, n=14) to clinical primary ovarian cancer samples from the microarray dataset GSE15622. There are 11 apoptosis/cell death–related pathways; 9 immune-related pathways; and 3 NF-κB-related pathways (I-κB kinase/NF-κB cascade)

**Supplementary Table 2**

Clinicopathological features of 21 ovarian cancer patients before and after chemotherapy at Kyoto University.

**Supplementary Table 3**

Frozen tumor specimens were obtained from ID8 tumor-bearing mice treated with GEM or PTX and a corresponding control group; mice were euthanized before reaching a moribund state. The intensity of the staining scores of NF-κB p65 or PD-L1 was detected and analyzed by immunohistochemistry using anti-NF-κB p65 or anti-PD-L1 antibodies (n=5).

**Supplementary Table 4**

Frozen tumor specimens were obtained from ID8 tumor-bearing mice treated with GEM or PTX and a corresponding control group; mice were euthanized before reaching a moribund state. The numbers of cells positive for CD8, CD4, or Gr-1 were determined by immunohistochemistry using anti-CD8, anti-CD4, or anti-Gr-1antibodies; original magnification 200× (n=5).

**Supplementary Figure 1. Upregulation of NF-κB p65 in mouse ovarian cancer cell lines following treatment with chemotherapeutic agents.**

(A) NF-κB p65 protein expression in mouse ovarian cancer cell lines (ID8 and HM-1) after CPA (0.7μM, 7 μM, 70 μM) treatment for 24 hours, analyzed by western blotting.

(B) NF-κB p65 protein expression in mouse ovarian cancer cell lines (ID8 and HM-1) after GEM (24 μM, 120 μM, 600 μM), PTX (2 μM, 10 μM, 20 μM), CBDCA (20 μM, 100 μM, 500 μM), or CPA (0.7μM, 7 μM, 70 μM) treatment for 24 hours, analyzed by western blotting. Densitometric analysis of NF-κB p65 was based on three independent experiments. \*, *P* < 0.05; \*\*, *P* < 0.01.

**Supplementary Figure 2. NF-κB p65 and PD-L1 expression in mouse ovarian cancer cell lines (ID8 and HM-1) after chemotherapeutic agents**

Full western blotting data from Figures 2A, 3D.

**Supplementary Figure 3. NF-κB p65, PD-L1 and MHC expression in human ovarian cancer cell lines (ovary1847 and SK-OV-3) after Gemcitabine or Paclitaxel**

Full western blotting data from Figures 2B, 2D, 3F.

**Supplementary Figure 4. Upregulation of MHC class I in ovarian cancer cell lines following treatment with chemotherapeutic agents.**

(A) MHC class I expression in mouse ovarian cancer cell lines (ID8 and HM-1) after GEM (120 μM), PTX (10 μM), or CBDCA (100 μM) treatment for 24 hours, analyzed by flow cytometry. Statistical analysis of PD-L1 was based on three independent experiments. \*, *P* < 0.05; \*\*, *P* < 0.01.

(B) MHC class I expression in human ovarian cancer cell lines (ovary1847, SK-OV-3) after GEM (24 μM, 120 μM, 600 μM) or PTX (2 μM, 10 μM, 20 μM) treatment for 24 hours, analyzed by western blotting. Densitometric analysis of MHC class I was based on three independent experiments. \*, *P* < 0.05; \*\*, *P* < 0.01.

(C) MHC class I expression in human ovarian cancer cell lines (RMGII, OVCAR8) after GEM (120 μM), PTX (10 μM) treatment for 24 hours, analyzed by western blotting. Densitometric analysis of MHC class I was based on three independent experiments. \*\*, *P* < 0.01.

**Supplementary Figure 5. Upregulation of PD-L1 mRNA expression in microarray dataset GSE18728.**

PD-L1 mRNA before and after docetaxel (DTX) plus capecitabine (CAPE) combination chemotherapy in breast cancer patients (n=14) from microarray dataset GSE18728.

**Supplementary Figure 6.Upregulation of PD-L1 in mouse ovarian cancer cell lines following chemotherapeutic treatment *in vitro.***

(A) ID8 and HM-1 cells were incubated with GEM (120 μM), PTX (10 μM), or CBDCA (100 μM) for 24 hours. Cell surface PD-L1 was detected by flow cytometry. Statistical analysis of PD-L1 was based on three independent experiments. \*\*, *P* < 0.01.

(B) ID8 and HM-1 cells were incubated with GEM (24 μM, 120 μM, 600 μM), PTX (2 μM, 10 μM, 20 μM), CBDCA (20 μM, 100 μM, 500 μM), or CPA (0.7μM, 7 μM, 70 μM) for 24 hours. Total cellular PD-L1 protein was measured by western blotting. Densitometric analysis of PD-L1 was based on three independent experiments. \*, *P* < 0.05; \*\*, *P* < 0.01.

(C) ID8 and HM-1 cells were incubated with CPA (0.7μM, 7 μM, 70 μM) for 24 hours. Total cellular PD-L1 protein was measured by western blotting.

(D) ID8 and HM-1 cells were incubated with IFN-γ (500pg/ml, 1ng/ml, 4ng/ml) for 24 hours. Total cellular PD-L1 protein was measured by western blotting.

**Supplementary Figure 7.Upregulation of PD-L1 in human ovarian cancer cell lines following chemotherapeutic treatment *in vitro.***

(A) Human ovarian cancer cell lines (RMGII, and OVCAR8) were cultured with GEM (120 μM) or PTX (10 μM) for 24 hours. Cell surface PD-L1 was detected by flow cytometry (red, isotype; green, anti-PD-L1 antibody).

(B) Human ovarian cancer cell lines (ovary1847, SK-OV-3, RMGII, and OVCAR8) were cultured with GEM (120 μM) or PTX (10 μM) for 24 hours. Cell surface PD-L1 was detected by flow cytometry. Statistical analysis of PD-L1 was based on three independent experiments. \*\*, *P* < 0.01.

(C) Human ovarian cancer cell lines (RMGII, and OVCAR8) were cultured with GEM (120 μM) or PTX (10 μM) for 24 hours. Total cellular PD-L1 was measured by western blotting. Densitometric analysis of PD-L1 was based on three independent experiments. \*\*, *P* < 0.01.

(D) PD-L1 expression in human ovarian cancer cell lines (ovary1847, SK-OV-3) after GEM (24 μM, 120 μM, 600 μM) or PTX (2 μM, 10 μM, 20 μM) treatment for 24 hours, analyzed by western blotting. Densitometric analysis of PD-L1 was based on three independent experiments. \*, *P* < 0.05; \*\*, *P* < 0.01.

**Supplementary Figure 8. Generation of NF-κB p65 knockdown mouse ovarian cancer cells with shNF-κB p65 (Rela knockdown).**

(A) Rela mRNA expression analyzed by RT-PCR in ID8 or HM-1 cells transduced with control shRNA or NF-κB p65 shRNA (two types of shRNA, rela69 and rela70). \*, *P* < 0.05.

(B) Total cellular NF-κB p65 was measured by western blotting in ID8 or HM-1 cells transduced with control shRNA or NF-κB p65 shRNA.

(C) Densitometric analysis of NF-κB p65 from western blotting was based on three independent experiments.\*, *P* < 0.05.

**Supplementary Figure 9.  Chemotherapeutic agents upregulate MHC class I via NF-**κ**B in mouse ovarian cancer cell lines *in vitro.***

Mouse MHC class I expression in ID8 cells transduced with shNF-κB p65 (Rela69) or shControl after culturing with GEM (120 μM) or PTX (10 μM) for 24 hours, analyzed by flow cytometry. Statistical analysis of MHC class I was based on three independent experiments. \*, *P* < 0.05; \*\*, *P* < 0.01.

**Supplementary Figure 10.  Chemotherapeutic agents upregulate MHC class I and PD-L1 via NF-**κ**B in human ovarian cancer cell lines *in vitro.***

(A) Human ovarian cancer cell lines (ovary1847, SK-OV-3 or OVCAR8) transfected with siNF-κB p65 or siControl, then cultured with GEM (120 μM) or PTX (10 μM) for 24 hours. Densitometric analysis of total cellular NF-κB p65 from western blotting was based on three independent experiments. \*\*, *P* < 0.01.

(B) NF-κB p65, MHC class I and PD-L1 in human ovarian cancer cell line OVCAR8 transfected with siNF-κB p65 or siControl after culturing with GEM (120 μM) or PTX (10 μM) for 24 hours, analyzed by western blotting.

(C) MHC class I in human ovarian cancer cell line OVCAR8 transfected with siNF-κB p65 or siControl after culturing with GEM (120 μM) or PTX (10 μM) for 24 hours, analyzed by western blotting. Densitometric analysis of MHC class I was based on three independent experiments. \*\*, *P* < 0.01.

(D) PD-L1 in human ovarian cancer cell line OVCAR8 transfected with siNF-κB p65 or siControl after culturing with GEM (120 μM) or PTX (10 μM) for 24 hours, analyzed by western blotting. Densitometric analysis of PD-L1 was based on three independent experiments. \*\*, *P* < 0.01.

**Supplementary Figure 11. PD-L1 was enhanced by Gemcitabine or paclitaxel via NF-kB signal in human ovarian cancer cell lines (ovary1847, SK-OV-3 and OVCAR8).**

Full western blotting data from Figure 4C, 4F.

**Supplementary Figure 12. Chemotherapeutic agents upregulate PD-L1 via NF-**κ**B in mouse ovarian cancer cell lines *in vitro.***

(A) ID8 or HM-1 cells transfected with control siRNA or NF-κB p65 siRNA, then treated with GEM (120μM) or PTX (10μM) for 24hours. Densitometric analysis of total cellular NF-κB p65 in western blotting was based on three independent experiments. \*\*, *P* < 0.01.

(B) Mouse PD-L1 expression in HM-1 cells transfected with siNF-κB p65 or siControl after culturing with GEM (120 μM) or PTX (10 μM) for 24 hours, analyzed by western blotting. Densitometric analysis of PD-L1 was based on three independent experiments. \*\*, *P* < 0.01.

(C) Mouse PD-L1 expression in HM-1 transduced with shNF-κB p65 (Rela69, 70) or shControl after culturing with GEM (120 μM) or PTX (10 μM) for 24 hours, analyzed by western blotting. Densitometric analysis of PD-L1 was based on three independent experiments. \*\*, *P* < 0.01.

**Supplementary Figure 13. PD-L1 was enhanced by Gemcitabine or paclitaxel via NF-kB signal in mouse ovarian cancer cell lines (ID8 and HM-1).**

Full western blotting data from Figure 4D.

**Supplementary Figure 14. PD-L1 was enhanced by Gemcitabine or paclitaxel via NF-kB signal in human ovarian cancer cell lines (ovary1847, SK-OV-3 and OVCAR8).**

Full western blotting data from Figure 4E.

**Supplementary Figure 15. Knockdown of Ifngr1 expression in ID8 cells.**

ID8 cells transfected with control shRNA or IFNGR1 shRNA and cell surface IFNGR1 was detected by flow cytometry. Results are from one of three independent experiments.

**Supplementary Figure 16. PD-L1 expression is upregulated by Gemcitabine or paclitaxel in the absence of the IFN-γ signaling pathway.**

Full western blotting data from Figure 5A.

**Supplementary Figure 17. The effect of chemotherapeutic drugs on the immunological microenvironment of mouse tumor tissue.**

(A) Left: frozen-section mouse tumor tissue analyzed by immunohistochemistry using anti-NF-κB p65 or anti-PD-L1 antibodies (original magnification 400×; scale bar, 100 μm); anti-CD8 or anti-CD4 antibodies (original magnification 200×; scale bar, 100 μm) from the ID8 tumor-bearing mice treated with vehicle (control) or GEM. Right; the staining score for NF-κB p65, PD-L1 and the number of CD8+ TILs, CD4+ TILs from the ID8 tumor-bearing mice treated with vehicle (control) or GEM (n=5 in each group). \*, *P* < 0.05; \*\*, *P* < 0.01.

(B) CD107a+ CTLs were detected by flow cytometry following co-incubation with Ovalbumin peptide-loaded (OVA-loaded) PTX-pretreated ID8-pdl1KO (24 hours; 10 μM), OVA-loaded PTX-pretreated ID8-control (24 hours; 10 μM), OVA-loaded ID8-control or non-OVA-loaded ID8-control (negative control). Results are based on three experiments. \*, *P* < 0.05; \*\*, *P* < 0.01.

**Supplementary Figure 18. Expression of PD-L1 and NF-κB p65 in ID8MirControl (control), ID8Mirpdl1 (pdl1-depleted), or ID8pdl1 (pdl1-overexpressing) cells incubated with GEM or PTX.**

Mouse ovarian cancer cell lines (ID8MirControl (control), ID8Mirpdl1 (pdl1 depleted), and ID8pdl1 (pdl1 overexpressing)) were cultured with GEM (120μM) or PTX (10μM) for 24hours.

(A) PD-L1 mRNA was measured by RT-PCR from triplicate determinants. \*\*, *P* <0.01.

(B) Total cellular PD-L1 and NF-κB p65 were measured by western blotting.

**Supplementary Figure 19. Chemotherapeutic drugs combined with PD-L1/PD-1 signaling blockade treatment in mouse model**

(A) Overall survival analysis of ID8MirControl (ID8-control), ID8Mirpdl1 (ID8-pdl1KO), or ID8pdl1overexpression (ID8-pdl1) tumor-bearing syngeneic mice treated with or without GEM (each group, n=12).

(B) Overall survival analysis of ID8OVA tumor-bearing syngeneic mice treated with GEM alone, anti-PD-1 antibody (αPD-1) alone, the combination of GEM and aPD-1, or saline and IgG intraperitoneal injection (control) (each group, n=12).

**Supplementary Figure 20. Generation of ID8OVA cells.**

(A) ID8OVA cells (two clones: ID8OVA1, ID8OVA2) were generated and confirmed by OVA protein expression measured by western blotting; the ID8lacz cell line was used as a negative control.

(B) Cytotoxicity assay of the OVA-manipulated ID8 cells. Means ±SD (n=3).  \*, *P* < 0.05.

(C) ID8OVA and ID8lacz cells were cultured with GEM (120μM) or PTX (10μM) for 24 hours; total cellular PD-L1 and NF-κB p65 were measured by western blotting.

(D) ID8OVA and ID8lacz cells were cultured with GEM (120μM) or PTX (10μM) for 24hours. PD-L1 mRNA was measured by qRT-PCR from triplicate experiments. \*\*, *P* < 0.01.

**Supplementary Figure 21. Anti-PD-1 antibody enhances the immune activation effect of PTX in a mouse ovarian cancer model**

(A-B) Percentage of CD8+(A) and CD4+(B) TILs infiltration of tumor was analyzed by flow cytometry in ID8OVA tumor-bearing syngeneic mice treated with PTX alone, anti-PD-1 antibody (αPD-1) alone, the combination of PTX and aPD-1, or saline and IgG intraperitoneal injection (control) (each group n=5). \*, *P* < 0.05.

(C) Percentage of total TILs that are PD-1+, analyzed by flow cytometry in ID8OVA tumor-bearing syngeneic mice treated with PTX alone, anti-PD-1 antibody (αPD-1) alone, the combination of PTX and aPD-1, or saline and IgG intraperitoneal injection (control) (each group, n=5). \*, *P* < 0.05; \*\*, *P* < 0.01.

**Supplementary Figure 22. The effect of GEM and PTX on Gr-1+ MDSCs.**

Left: immunohistochemistry analysis of frozen-section mouse tumor tissue using anti-Gr-1 antibody (original magnification 200×; arrow, Gr-1+cells; scale bar, 100μm) from the ID8 tumor-bearing mice PTX-treated group, GEM-treated group and corresponding control group. Right: the number of Gr-1+cells were counted in untreated (control), PTX-treated group, or GEM-treated group (n=5). \*, *P* < 0.05.