Figure S1. OPNc knockdown modulates the mRNA expression of the OPNa and OPNb isoforms. Cisplatin-resistant ACRP ovarian cancer cells were transfected with ASO SCR or ASO anti-OPNc, and reverse transcription-quantitative PCR was used to analyze OPNa and OPNb mRNA expression levels. Log_{10} relative expression levels were calculated using the $2^{-\Delta\Delta Cq}$ method and normalized to those of β-actin. ns, non-significant vs. ASO SCR. OPN, osteopontin; ASO, antisense oligonucleotide; SCR, scramble.

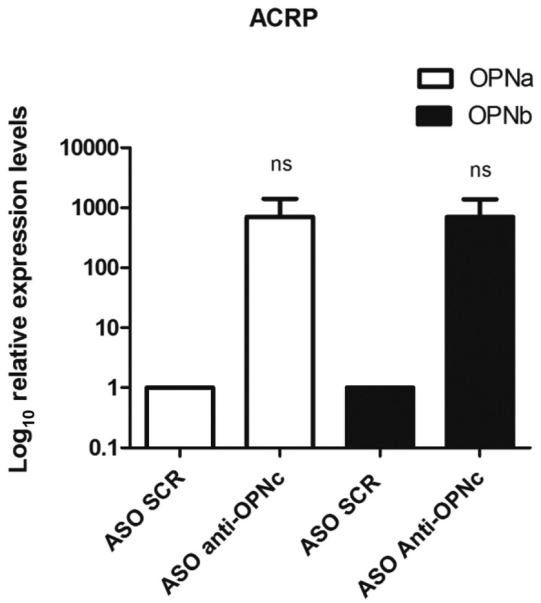

Figure S2. OPNc knockdown sensitizes OVCar-8 Dox^R cells to treatment and impairs cell viability. (A and B) OVCar-8 Dox^R ovarian cancer cells were transfected with ASO SCR or ASO anti-OPNc, and reverse transcription-quantitative PCR was used to analyze the mRNA expression levels of (A) OPNc and (B) P-gp. Relative expression levels were calculated using the $2^{-\Delta\Delta Cq}$ method and normalized to those of β-actin. (C and D) Following 24-h transfection, OVCar-8 Dox^R cells were treated with increasing concentrations of doxorubicin for (C) 24 and (D) 96 h. Cell viability was measured by the MTT assay. $^*P\leq0.05$, $^{**}P\leq0.01$ and $^{***}P\leq0.001$; ns, non-significant. OPNc, osteopontin-c isoform; Dox^R, doxorubicin-resistant; ASO, antisense oligonucleotide; SCR, scramble; P-gp, P-glycoprotein.

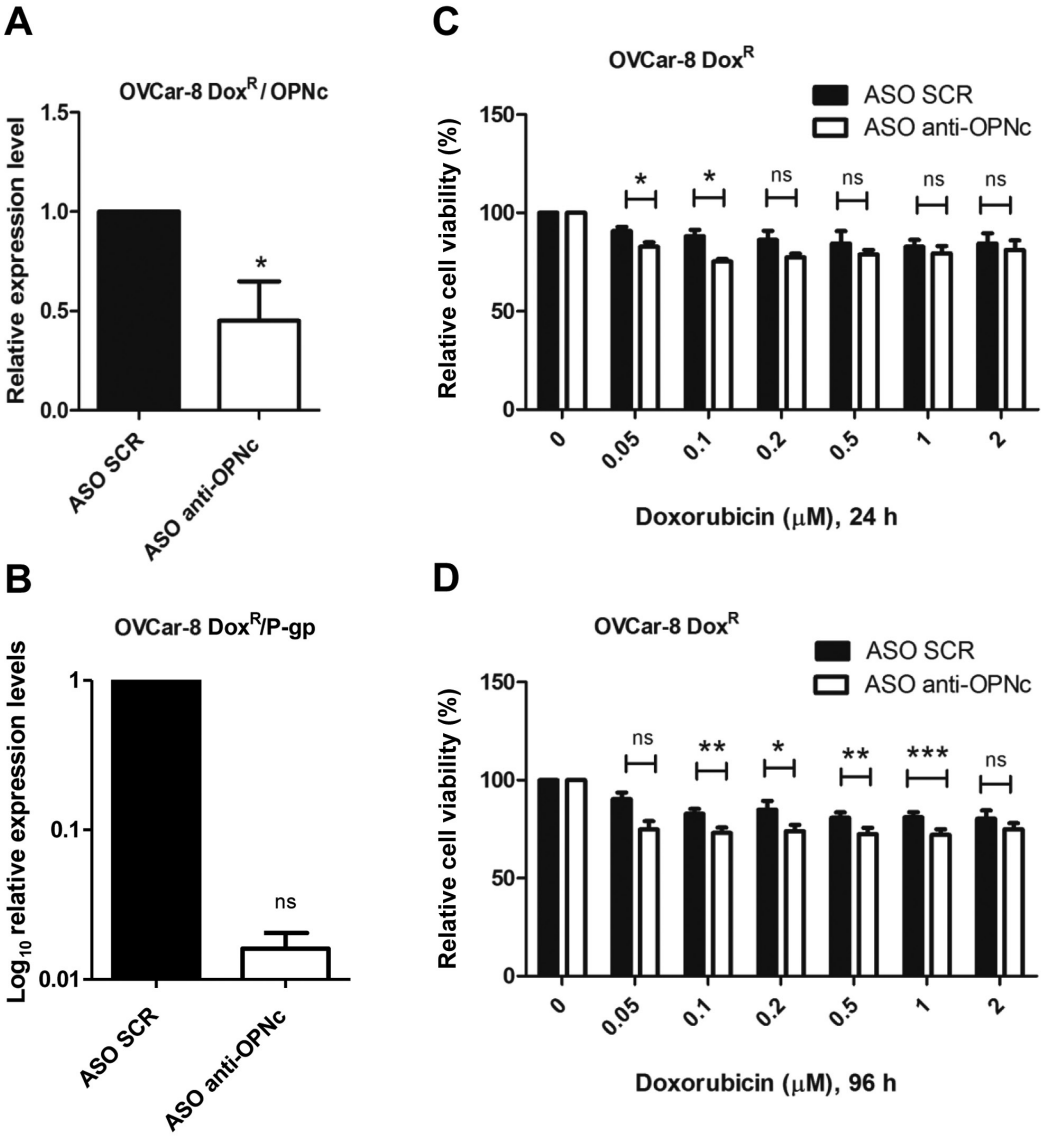

Figure S3. OPNc knockdown impairs the viability of OVCar-8 Dox^R cells. (A) OVCar-8 Dox^R ovarian cancer cells were transfected with ASO SCR or ASO anti-OPNc, and cell viability was determined using trypan blue exclusion assay. (B) Clone formation ability of OPNc-silenced OVCar-8 Dox^R cells was assessed after 14-day incubation by crystal violet staining. Optical density was obtained at 595 nm. $^*P \leq 0.05$; ns, non-significant. OPNc, osteopontin-c isoform; DoxR, doxorubicin-resistant; ASO, antisense oligonucleotide; SCR, scramble.

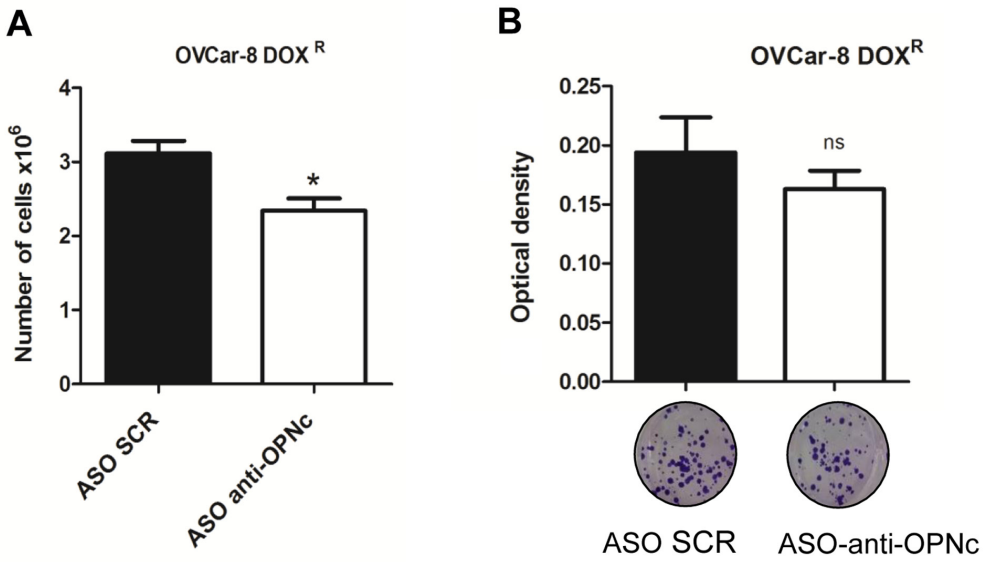

Figure S4. OPNc regulates the expression of epithelial-mesenchymal transition-related cytokines in doxorubicin-resistant cancer cells. OVCar-8 Dox^R cells were transfected with ASO SCR or ASO anti-OPNc, and the mRNA levels of IL-6, IL-8, IL-1α, IL-1β and the GP130 receptor were analyzed by reverse transcription-quantitative PCR. The levels of mRNA were normalized those of β-actin. $^{*}P\leq0.05$, $^{**}P\leq0.01$ and $^{***}P\leq0.001$; ns, non-significant. OPNc, osteopontin-c isoform; Dox^R, doxorubicin-resistant; ASO, antisense oligonucleotide; SCR, scramble; GP130, glycoprotein 130.

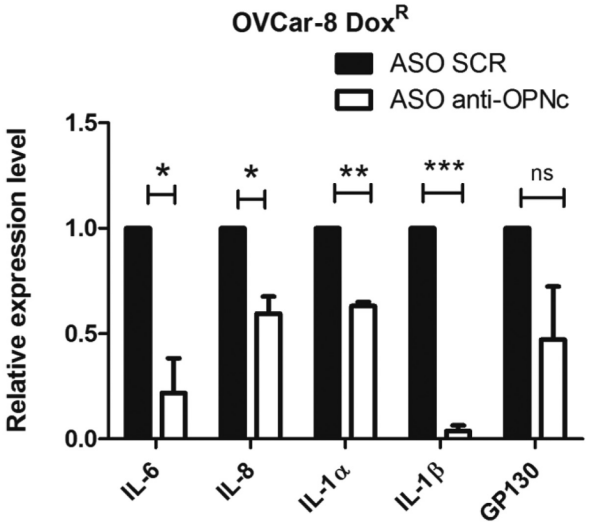

Figure S5. OPNc overexpressing cells exhibit attenuated chemosensitivity and OPNc modulation following 96-h cisplatin treatment. (A) A2780 OPNc$^+$ and pCR3.1-transfected cells were treated with increasing concentrations of cisplatin for 96 h, and cell viability was measured by the MTT assay. (B) A2780 OPNc$^+$ and pCR3.1-transfected were treated with 10 μM cisplatin for 96 h, and OPNc expression levels were analyzed by reverse transcription-quantitative PCR. Log10 relative expression levels were calculated using the $2^{-\Delta\Delta Cq}$ method and normalized to those of β-actin. ns, non-significant. OPNc, osteopontin-c isoform; OPNc$^+$, OPNc-overexpressing; pCR3.1, empty vector.