

Supplementary Fig. S1. Chemokines CXCL7 and chemerin exert no remarkable impact on activation of TAK1/NF κ B signalling in ovarian cancer cells. (A) XTT cell proliferation assay demonstrated that cell growth of SKOV3 cells was not influenced by the treatment of CXCL7 (10 and 20 ng/mL). Western blot analysis further confirmed that CXCL7 (1.25, 2.5, 5, 10 and 20 ng/mL) could not provoke the phosphorylation of TAK1 and its downstream targets IKK α / β and I κ B α in SKOV3 cells. (B) XTT cell proliferation assay showed that cell growth of A2780cp cells was not promoted by the treatment of chemerin (20 ng/mL). Western blot analysis further strengthened that insignificant effect on the phosphorylation of TAK1 and its downstream targets targets IKK α / β and I κ B α was observed in A2780cp cells upon treatment of chemerin (1.25, 2.5, 5, 10 and 20 ng/mL) for 24 h.



Supplementary Fig. S2. IL-8 but not GRO- α exerts positive feedback loop in SKOV3 cells. QPCR analysis revealed that treatment of IL-8 (25 and 50 ng/mL) but not GRO- α (20, 40 and 80 ng/mL) for 24 h, induced a positive feedback loop in SKOV3 cells dose dependently. But co-treatment of CXCR2 inhibitor SB225002 (5 μ M) or TAK1 inhibitor (5z)-7-oxozeaenol (2.5 μ M) could remarkably inhibit the induction of IL-8 in SKOV3 when co-treated with IL-8 (50 ng/mL) for 24 h.

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Supplementary Fig. S3. NF κ B signalling is a direct transcriptional target of GRO- α /IL-8 and OCM. (A) Luciferase reporter assay using NF κ B promoter luciferase reporter construct (luc- NF κ B) demonstrated that GRO- α (20, 40 and 80 ng/mL) and IL-8 (20 and 40 ng/mL) elevate the luciferase reporter signals from ~10 to 25% and from ~50 to 60% respectively in HEK293 cells upon 48 h incubation. (B) Using luc-NF κ B and co-treatment with anti-GRO- α , anti-IL-8, anti-CXCR2 antibodies (2 µg/mL) or (5z)-7-oxozeaenol (2.5 µM) in HEK293 cells for 48 h, the chemokines-upregulated NF κ B signals were remarkably reduced at least 50%.

В

CXCR2



Supplementary Fig. S4. Correlation between CXCR2 expression and 5-year survival rates of ovarian cancer patients. Kaplan–Meier analyses of 5-year progressive free survival (PFS, *left panel*), post progression survival (PPS, *middle panel*) and overall survival (OS, *right panel*) rate based on clinical and molecular data for ovarian cancer patients (n = 1305 for PFS, n = 1581 for PPS and n = 707 for OS). Cut-off value was auto-selected by the software to stratify patients into low and high *CXCR2* expression groups. Log-rank (Mantel-Cox), *P* values and hazard ratios (HRs; 95% confidence interval in parentheses) were presented.



Supplementary Fig. S5. Knockout of CXCR2 on SKOV3 cells exhibited lower cancer cell aggressiveness upon chemokines treatment. Transwell migration assay showed that SKOV3 scrambled control cells treated with GRO- α (20, 40 and 80 ng/mL) and IL-8 (25 and 50 ng/mL) for 18 h exhibited a dose-dependent upsurge in the number of cells penetrating through the membrane as compared with untreated control (**P* < 0.05). The GRO- α and IL-8-stimulated cell migration in SKOV3 *CXCR2*^{-/-} cells was successfully abolished by knocking out CXCR2 (* *P* < 0.05), although there was still a little increment of cell migration at high concentration of GRO- α (80 ng/mL). Numbers of migrated cells in three randomly chosen fields were counted for three independent experiments and the normalized numerical data were presented in bar charts with error bars. Scale bar = 50µm.