

Supplementary data

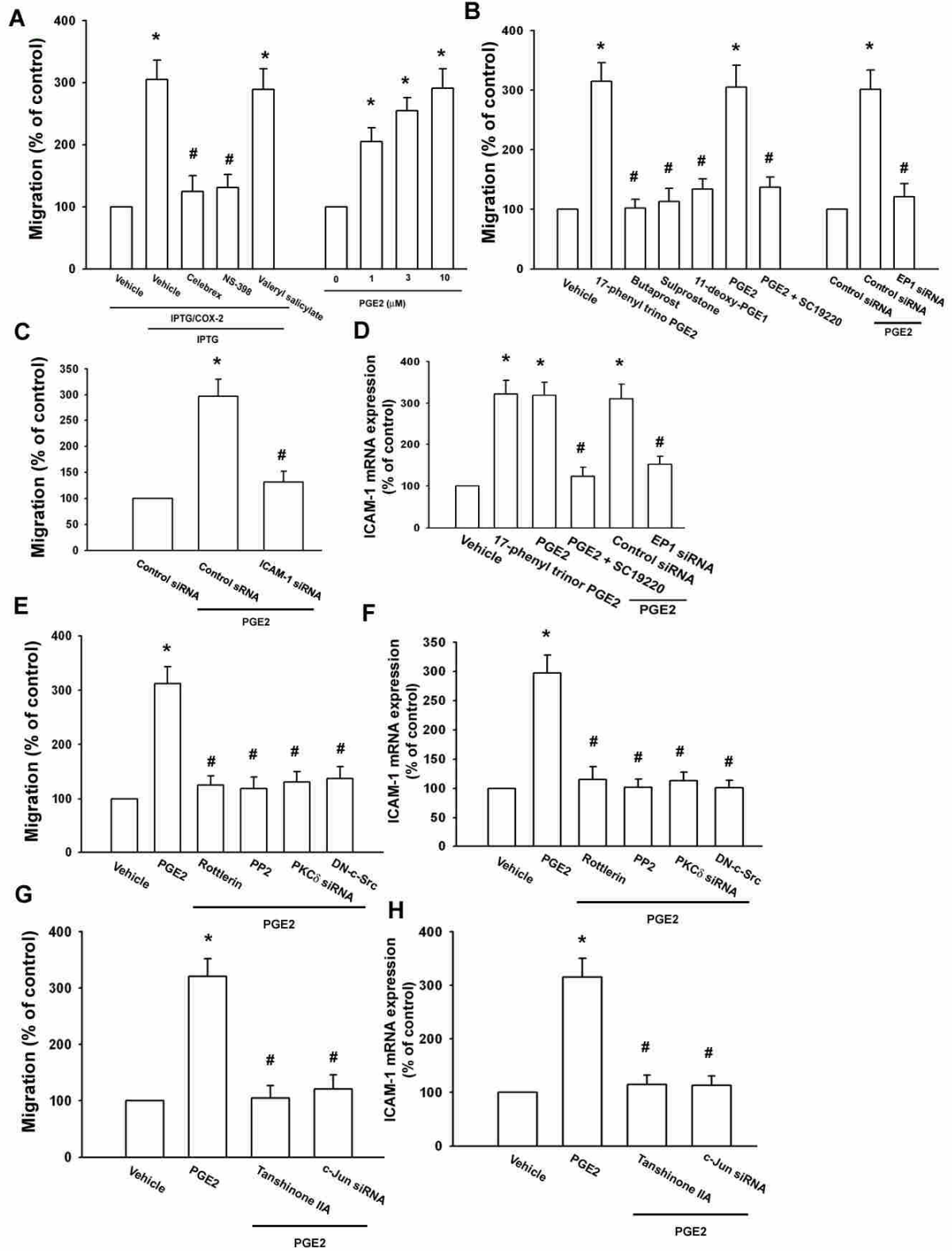


Fig. S1. PGE₂ induced cell migration and ICAM-1 expression through EP1, PKC δ , c-Src and AP-1 signaling pathways in human oral cancer HSC3 cells.

(A) HSC3 cells were transfected with IPTG/COX-2 expression plasmid or control vector for 24 hr, and pretreated with valeryl salicylate (20 μ M), celebrex (10 μ M) or NS-398 (20 μ M) for 30 min followed by stimulation with IPTG (5 mM), and *in vitro* migration was measured with the Transwell after 24 hr. (A; right part) HSC3 cells were incubated with various concentrations of PGE₂, and *in vitro* migration activity measured with the Transwell after 24 hr.

(B) HSC3 cells were treated with 17-phenyl trinor PGE₂ (3 μ M), butaprost (10 μ M), sulprostone (10 μ M), 11-deoxy-PGE₁ (10 μ M), PGE₂ and PGE₂ plus SC19220 (10 μ M), and *in vitro* migration activity measured with the Transwell after 24 hr. (B; right part) HSC3 cells were transfected with EP1 siRNA for 24 hr followed by stimulation with PGE₂, and *in vitro* migration measured with the Transwell after 24 hr.

(C) HSC3 cells were transfected with ICAM-1 siRNA for 24 hr followed by stimulation with PGE₂, and *in vitro* migration measured with the Transwell after 24 hr.

(D) HSC3 cells were treated with 17-phenyl trinor PGE₂ (3 μ M), 11-deoxy-PGE₁ (10 μ M), PGE₂, and PGE₂ plus SC19220 (10 μ M), and mRNA expression of ICAM-1 was determined using qPCR.

HSC3 cells were pretreated for 30 min with rottlerin and PP2 or transfected with PKC δ siRNA and c-Src mutant for 24 hr followed by stimulation with PGE₂ for 24 hr, and *in vitro* migration (E) and ICAM-1 expression (F) were measured with the Transwell and qPCR after 24 hr.

HSC3 cells were pretreated for 30 min with tanshinone IIA or transfected with c-Jun siRNA for 24 hr followed by stimulation with PGE₂ for 24 hr, and *in vitro* migration (G) and ICAM-1 expression (H) were measured with the Transwell and qPCR after 24 hr.

Results are expressed as the mean \pm S.E. *: $p < 0.05$ as compared with control. #: $p < 0.05$ as compared with PGE₂-treated group

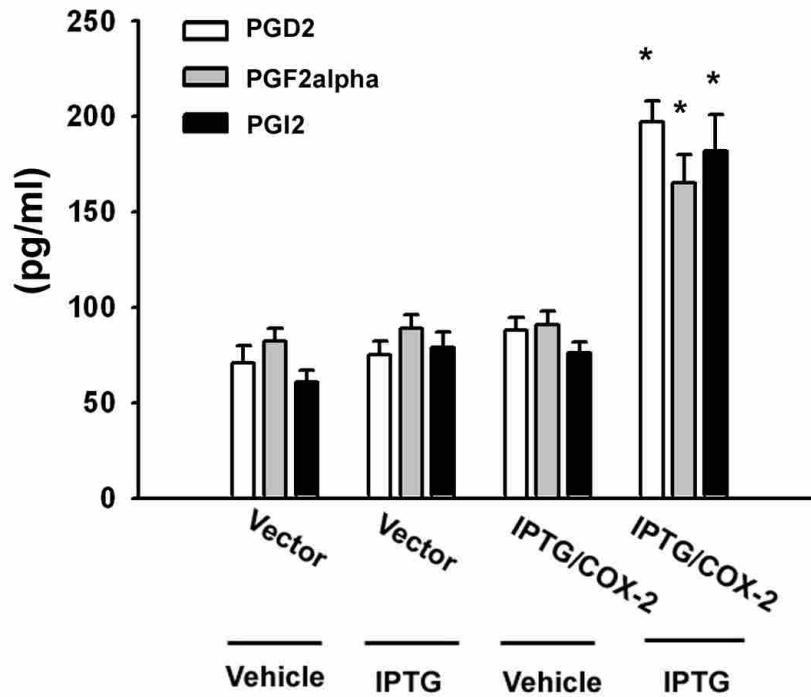


Fig. S2. COX-2 increased PGD2, PGF2alpha and PGI2 production in human oral cancer cells.

SCC4 cells were transfected with IPTG/COX-2 expression plasmid or control vector for 24 hr followed by stimulation with IPTG (5 mM) for 24 hr, the PGD2, PGF2alpha and PGI2 production were determined by ELISA assay. Results are expressed as the mean \pm S.E. *, $p < 0.05$ compared with control.

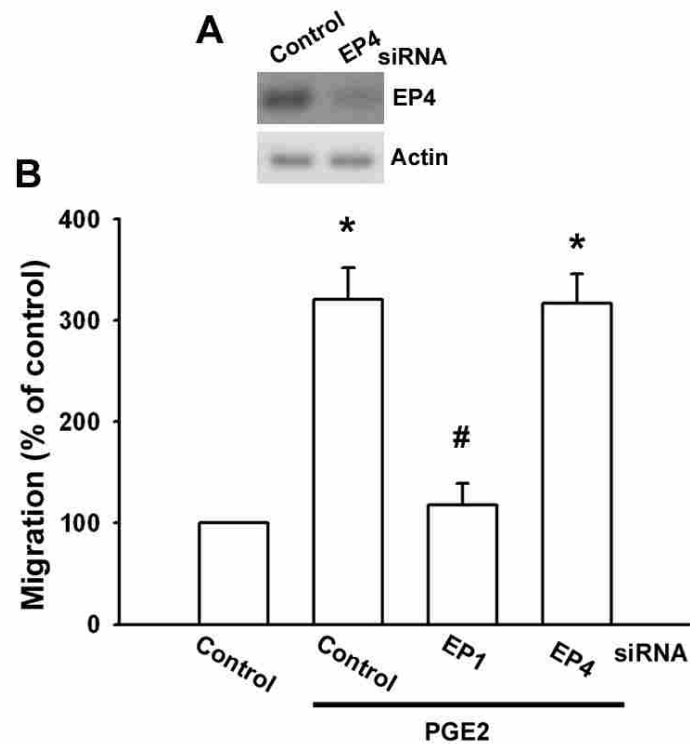


Fig. S3. EP4 receptor did not affect PGE₂-induced cell migration in human oral cancer cells.

(A) SCC4 cells were transfected with control or EP4 siRNA for 24 hr, and EP4 expression was examined by Western blot. (B) SCC4 cells were transfected with control, EP1 or EP4 siRNA for 24 hr followed by stimulation with PGE₂ for 24 hr, and *in vitro* migration were measured with the Transwell after 24 hr. Results are expressed as the mean \pm S.E. *: $p < 0.05$ as compared with control. #: $p < 0.05$ as compared with PGE₂-treated group

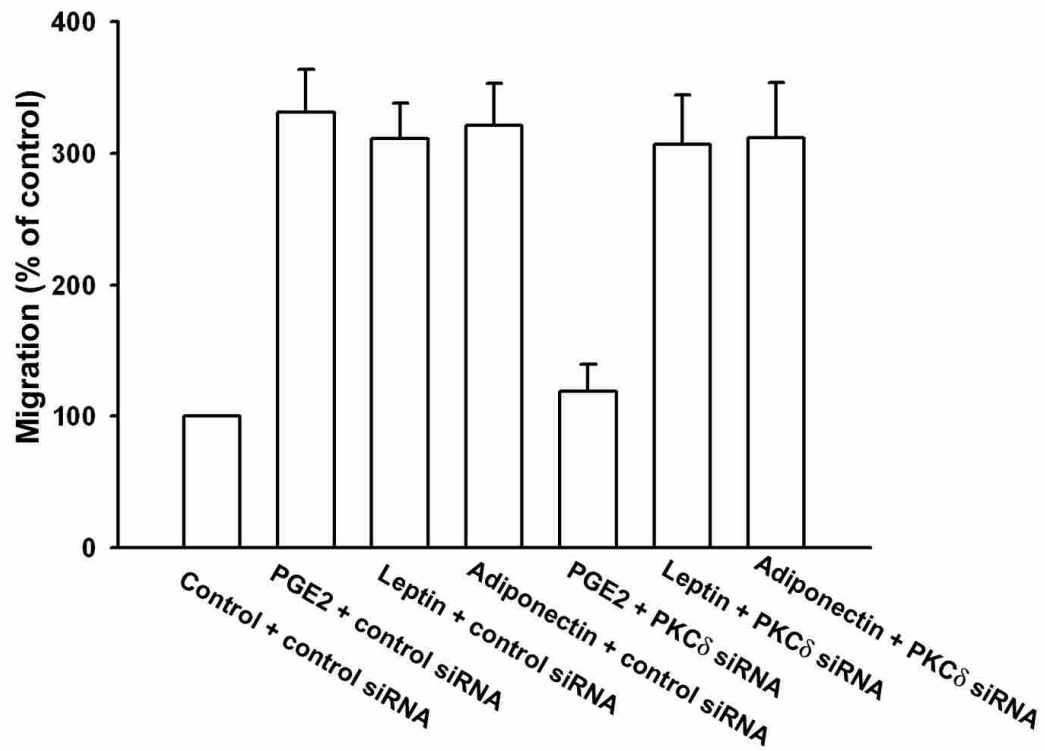


Fig. S4. Leptin or adiponectin induce cell migration through PKC δ -independent pathway in human oral cancer cells.

SCC4 cells were transfected with control or PKC δ siRNA for 24 hr followed by stimulation with PGE₂, leptin or adiponectin for 24 hr, and *in vitro* migration were measured with the Transwell after 24 hr.