**Supplementary Figure Legends**

**Supplementary Figure S1. After EGF (10 ng/ml) treatment, Cdc42 and Rac1 were maximally activated in SKOV3ip cells after 5 and 20 min growth factor stimulation, respectively.** SKOV3ip cells were cultured overnight to reach ~80% confluence, then stimulated with 10 ng/ml EGF for different time length (0 - 40 min). The activation was terminated by washing with ice cold PBS. Cells were lysed with RIPA buffer and the activated GTPase levels were measured using a flow cytometry based GTPase effector binding assay. Normalized activities are expressed relative to unstimulated controls.

**Supplementary Figure S2.** **Cell characterization of primary ovarian cancer cells by immunofluorescence.** (A) Cells were purified via Ficoll gradient and negative selection using CD45-Dynabeads. Cells were stained for CA125 and EpCAM (clone Ber-EP4). (B) Isolated cells were expanded by cell culture and stained for CA125 and EpCAM (clone Ber-EP4).

**Supplementary Figure S3. Ketorolac inhibition of p-PAK1 (Thr423)/PAK2(Thr402) and p-PAK1(Ser199)/PAK2(Ser192).** (A) A model for PAK1 activation. Based on the published structure ([42](#_ENREF_42)), PAK1 contains a N-terminal regulatory domain, including Cdc42/Rac1 interaction/binding (CRIB) and auto-inhibitory domain (AID), a PAK-interacting exchange factor (PIX) and a kinase domain. In inactive state, the PAK is auto-inhibited through the formation of dimers between the AID and the kinase domain to prevent auto-phosphorylation. In the present of GTP-Cdc42/Rac1, the GTPase CRIB complex catalyzes phosphorylation of Serine144 at AID, which disables the AID-kinase domain interaction and activates the monomeric PAK auto-phosphorylation kinase domain at Threonine 423. The phosphorylation of Serine 199 is activated by a PAK-interacting exchange factor (PIX). (B-E) Quantification of three independent measurements of the expression of p-PAK1(Thr423)/PAK2(Thr402) and p-PAK1(Ser199)/PAK2(Ser192) levels in SKOV3ip cells +/- drug treatments as indicated. . p-PAK1/PAK2 levels were normalized to unstimulated controls.