Supplementary figure captions

Figure S1. Arginase does not regulate arginine levels in OVCAs and ECAs

(A) Urea levels of OVCAs and ECAs were measured in the spent media using Urea Nitrogen (BUN) Reagent Set (purchase from Pointe Scientific) according to the manufacturer’s protocol. L-arginase (10U/mL) was used as the positive control in fresh medium. (B) Comparison of arginase-1 (ARG1) expression levels between normal ovarian surface epithelium and ovarian and endometrial adenocarcinomas. Data were downloaded from Oncomine. Data are expressed for each cell type as the mean ± SE; n>6. *P<0.05, §P<0.01, and #P<0.001. T-test was used for single comparisons.

Figure S2. O-ASCs induce reduced viability of cancer cells deprived of arginine.

(A) Cancer media (RPMI 1640 without arginine) was conditioned with O-ASCs for 24 hours. Cancer cells were cultured in conditioned media for three days while the media was changed every other day. Cancer cells cultured without arginine was used as control condition. (B) O-ASC 10 and IMR-90 were seeded on 96-well plate and cultured with and without arginine. Viability of cells was measured using CellTiter 96® AQueous Non-Radioactive Cell Proliferation Assay (purchased from Promega) according to manufacturer’s protocol. Data are expressed as means ± SE; n>6. *P<0.05, §P<0.01, and #P<0.001. T-test was used for single comparisons. Multiple comparisons versus a control group were analyzed by Dunnett's method. All pairwise multiple comparisons were analyzed by Bonferroni test to compare lean and obese patients’ samples.

Figure S3. L-arginase depletes arginine within the media

Complete RPMI was treated with different concentrations of L-arginase and the arginine content of the media was measured using UPLC.
**Figure S4.** Cancer cells induce glucose uptake and lactate secretion of O-ASCs and vice versa. OVCAR429 and HEC-1-A were transwell cocultured with O-ASCs for three days. The media was RPMI 1640 without arginine during three days of indirect coculture. Cocultured media was collected on the third day and diluted (1:1) with fresh RPMI (without arginine). Transwell inserts containing O-ASCs from coculture were separated from cancer cells and were placed in 6-well plate. Cancer cells and O-ASCs were separately cultured with diluted media for 24 hours prior to supernatant collection. Collected samples were analyzed for their extracellular metabolites content. (A) Results show pyruvate uptake of HEC-1-A, OVCAR429. Cancer cells and O-ASCs without coculture were used as controls. (B) and (C) Glucose uptake, lactate secretion and pyruvate uptake are shown, respectively for O-ASCs with and without coculture of O-ASCs. O-ASCs without coculture were used as the control. Data are expressed for each cell type as the mean ± SE; n>3. *P<0.05, §P<0.01, and #P<0.001. Dunnett's method was used to compare multiple groups versus a control group.

**Figure S5.** NO induces resistance of cancer cells to paclitaxel.

(A) HEC-1-A and OVCAR429 were seeded in 96-well plate and treated with paclitaxel (500nM), L-NAME (20mM), SNAP (100nM), and L-arginase (10U/ml) or combinations thereof. W/O ARG labeling denotes cancer media, RPMI 1640, without arginine. Viable cancer cells were determined by measuring luminesces of luciferin (150μg/mL) conversion after 72 hours. Data are expressed as means ± SE; n>6. *P<0.05, §P<0.01, and #P<0.001. All pairwise multiple comparisons were analyzed by Bonferroni test.