Supplementary Figure 1. ARID1A loss in the OSE does not lead to tumor formation. (A) Survival curves for two large cohorts of AdCRE injected Arid1a^{fl/fl} mice aged approximately one year. The mice remained tumor-free and no significant changes in survival were found throughout this extended observation period. Age-related morbidity issues were detected in the mice, resulting in euthanasia. (B,C) Representative histological sections of control (uninjected) or AdCRE exposed ovaries showing no evidence of tumor formation.
Supplementary Figure 2. SNP array analysis of Arid1Afl/fl (Gt)Rosa26Pik3caH1047R ovarian tumors and metastases. DNA from 12 tumor samples, consisting of matched primary tumors and two metastases, from 4 mice was analyzed using a high density mouse SNP array. Each tumor DNA set was matched with normal DNA from the same mouse. Shown are ASCAT profiles of genome-wide copy number changes in two primary tumors and matched metastases. Green and red bars demarcate the allele A and B, respectively. Chromosome-wide copy number variation was not detected in the tumors. Other low-level changes are consistent with normal background variation.
Supplementary Figure 3. OSE hyperplasia and tumor cell exfoliation occurs within two weeks after AdCRE injection in Arid1a^{fl/fl}; (Gt)Rosa26Pik3ca^{H1047R} mice. (E,F,H) Hobnail cells (arrowheads) are observed budding from the OSE one week post-AdCRE injection. OSE hyperplasia and exfoliating cell aggregates combined with hobnail cells are observed two weeks post-AdCRE injection. (F,H, arrows) Clear cell differentiation is observed in tumor forming regions at two weeks post-AdCRE. (G) Hyperplastic cells and early exfoliated cell aggregates expressed the OSE marker, cytokeratin 8 (CK8). Oo, oocyte; OV, ovary.
Supplementary Figure 4. Statistical analysis of Arid1a^{fl/fl};(Gt)Rosa26Pik3ca^{H1047R} ovarian tumor versus normal ovary microarrays. (A) Heat map of differentially expressed genes identified through SAM analysis (FDR=0). (B) PIK3CA and ARID1A expression in normal control ovaries and primary tumors.
Supplementary Figure 5. RT-PCR validation of *Arid1a*ββ*<sup>−/−</sup>*(Gt)*Rosa26Pik3ca*<sup>+</sup>*H1047R* ovarian tumor gene expression. (A) Heat map of a subset of differentially expressed genes that are commonly upregulated in human OCCC. (B) RT-PCR validation of microarray experiments in control, normal ovary, primary tumor, and peritoneal metastases.
Supplementary Figure 6. Cross-species gene expression comparisons using control ovary samples. Clustered heatmap of cross-species differential gene expression. 584 genes (272, upregulated; 312, downregulated) were coordinately altered in both mouse and human OCCC tumors (tumor vs. species-matched control ovary). (B) Top six Ingenuity Pathway Analysis (IPA) and GSEA Molecular Signatures Database (MSigDB) predictions of genes upregulated in mouse and human OCCC vs. species-matched control ovaries.
| Supplementary Figure 7. List of all coordinately regulated genes in mouse tumors and human OCCC (tumor versus control ovary). |
Supplementary Figure 8. Principal component analysis (PCA) plot of Arid1a$^{fl/fl};(Gt)Rosa26Pik3ca$T1047R and Arid1a$^{fl/fl};PTEN$null tumor gene expression.
Supplementary Figure 9. Marked up images of raw western blot scans. Crop marks are approximated by dashed lines. Each scan is labeled with the corresponding figure panel designation found in the main article figures or text.
Supplementary Figure 10. Marked up images of raw western blot scans. Crop marks are approximated by dashed lines. Each scan is labeled with the corresponding figure panel designation found in the main article figures or text.