Figure S1. Kaplan-Meier survival curves for overall survival according to chromatin remodeling factor mRNA expression of patients with ovarian serous carcinoma, using data compiled from the cBioPortal based on original data from The Cancer Genome Atlas. The threshold value between the high and low groups was determined by the median normalized RNA-seq by expectation maximization values. (A) *ACTL6A*, (B) *SMARCC2* and (C) *CHD4* mRNA expression levels were not significantly associated with OS. (D) *ACTL6A* mRNA expression was higher in patients with *ACTL6A* DNA copy number amplification compared with those with unaltered *ACTL6A*. RSEM, RNA-seq by expectation maximization; *ACTL6A*, actin-like protein 6A; *SMARCC2*, switch/sucrose non-fermentable related, matrix associated, actin dependent regulator of chromatin subfamily c member 2; *CHD4*, chromodomain-helicase-DNA-binding protein 4.



Figure S2. Representative immunohistochemistry images of (A) ARID1A, (B) SMARCA2, (C) SMARCA4, (D) SMARCB1, (E) SMARCC2 and (F) H3K27me3 in ovarian high-grade serous carcinoma. Positive staining was detected in the nucleus of tumor cells and endothelium of blood vessels. Scale bar, 50  $\mu$ m. ARID1A, AT-rich interaction domain 1A; SMARC, switch/ sucrose non-fermentable related, matrix associated, actin dependent regulator of chromatin; H3K27me3, tri-methylation of lysine 27 of histone H3.



Figure S3. Representative immunohistochemistry images of (A and B) ACTL6A, (C and D) SMARCC2 and (E and F) CHD4 in ovarian high-grade serous carcinoma. (A, C and E) High and (B, D and F) low nuclear expression of ACTL6A, SMARCC2 and CHD4 was observed in tumor cells. The nucleus of blood vessel endothelium also shows weak positive staining for these markers, serving as an internal positive control. ACTL6A, actin-like protein 6A; SMARCC2, switch/sucrose non-fermentable related, matrix associated, actin dependent regulator of chromatin subfamily c member 2; CHD4, chromodomain-helicase-DNA-binding protein 4.



Figure S4. Relationship between chromatin remodeling factor copy number, OS and FIGO stages in patients with OHGSC. Kaplan-Meier survival analysis was conducted for patients with (A) *ACTL6A*, (B) *SMARCC2* and (C) *CHD4* copy number gain or gene amplification, and shallow deletion or deep deletion. However, no statistically significant association was demonstrated between the CNA of *ACTL6A*, *SMARCC2* and *CHD4* and OS. The relationship between (D) *ACTL6A*, (E) *SMARCC2* and (F) *CHD4* copy numbers and the FIGO stages. No statistically significant association was demonstrated between the CNA of *ACTL6A*, *SMARCC2* and *CHD4* and the different FIGO stages. (G and H) Kaplan-Meier survival curves for OS according to patients with FIGO stage III/IV with CNAs. Patients with (G) copy number gain or amplification and (H) shallow or deep deletion in either *ACTL6A*, *SMARCC2* or *CHD4* demonstrated unfavorable outcome trends compared with those with all of the *ACTL6A*, *SMARCC2* and *CHD4* diploids, although this was not statistically significant. OS, overall survival; OHGSC, ovarian high-grade serous carcinoma; FIGO, International Federation of Gynecology and Obstetrics; CNA, copy number alteration; *CHD4*, chromodomain-helicase-DNA-binding protein 4; *ACTL6A*, actin-like protein 6A; *SMARCC2*, switch/sucrose non-fermentable related, matrix associated, actin dependent regulator of chromatin subfamily c member 2.

