**Description of Additional Supplementary Files**

**File Name**: Supplementary Data 1

**Description**: *RAD51C* ovarian cancer variants sourced from the literature, cancer databases, and/or our clinical collaborations are shown. Variants analyzed in this study are indicated with black text. Nucleotide change and mode of inheritance (germline, somatic, loss-of-heterozygosity (LOH) are indicated. In some cases, the RAD51C variant was identified in other cancer types (COAD, colon cancer; BRCA, breast cancer; LUAD, lung adenocarcinoma; BLCA, bladder cancer; LUSC, lung squamous cell carcinoma; KIRC, kidney cancer; PRAD, prostate cancer; ESCA, esophageal cancer; HCC, liver cancer; UEC, uterine cancer; SKCM, skin cancer). If the variant was identified in ClinVar or as a SNP on the gnomAD database, the corresponding reference number and SNP occurrence (Occur.) is indicated. Allele count is shown as well as any previous characterization of gray text variants and the literature reference.

**File Name**: Supplementary Data 2

**Description**: RAD51C variant viability complementation as determined by Olvera-León et al., interaction with RAD51D by yeast-three-hybrid (Y3H) relative to WT (%; **Fig. 1c,d**), HR relative to WT (%; **Fig. 2b**), sensitivity to cisplatin or olaparib (**Fig. 5**), ATPase activity (**Fig. 4e,f**), DNA binding activity (**Fig. 4c,d**), complex formation, and fork reversal proficiency (**Fig. 4a,b,g**) are summarized. [#]RAD51C-V239G drug sensitivity results were not statistically different from WT.