**SUPPLEMENTARY DATA**

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# 1. SUPPLEMENTARY METHODS

## 1.1 Tissue microarray immunohistochemistry

Hematoxylin & eosin (H&E) stained sections of formalin-fixed paraffin embedded tumor blocks were reviewed by a pathologist to select representative areas of tumor for coring. Duplicate cores (either 1 mm or 0.6 mm in diameter) were taken from each donor block and assembled in a recipient paraffin block using a manual precision instrument (Advanced Tissue Arrayer, ATA-100, Chemicon International). Sections of 4 μm thickness were mounted on Superfrost plus microscope slides and dried at 37°C for 72 hours. Immunohistochemical analysis for the expression of p53, WT1, CDKN2A/p16 and MIB1/Ki67 were performed on a Leica Bond III, for RB1 on a DAKO Omnis platform, and for CD8 on the Intellipath FLX autostaining system. Tissue microarray sections were pre-treated with the on board heat induced antigen retrieval, then incubated with each primary antibody at a given dilution. Details and explanation of interpretation are provided in Supplementary Table S2.

To score CD8+ cells, slides were scanned at 20x with a Vectra imaging system and analyzed using inForm Cell Analysis software (both PerkinElmer, Waltham, USA). Tumor epithelium and stromal regions were segregated using the tissue segmentation function of the software. Cells were segmented, phenotyped and counted automatically, with manual checking and correction. Tissue was included for analysis if total tissue surface was above threshold i.e. ≥ 20% of tumor epithelium (for TE) as well as ≥ 20% stroma (for STR) per 6 mm core, or the equivalent amount on a 1 mm core.

For markers with quantitative measurements (Ki-67 and CD8), scores are the average of 1 to 5 cores per case, and approximately 70% of cases have data from more than 1 core. Cases with at least two cores were tested for consistency in scoring between cores, and scores were significantly correlated for each quantitative marker (Ki-67, *P* < 0.001, *n* = 332; CD8 TE, *P* < 0.001, *n* = 291; CD8 STR, *P* < 0.001, *n* = 236; Spearman correlation *P* value reported). For continuous variables (Ki-67, CD8), the cut-off for “high” was set as ≥ top quartile of the control cohort (unselected HGSC).

## 1.2 DNA sequence analysis

## 1.2.1 DNA isolation

Fresh-frozen and archival formalin-fixed paraffin embedded tumor tissue samples collected at primary surgery were obtained from AOCS and GynBiobank, for participants with specimens available (Supplementary Table S1). For tumor samples containing at least 70% tumor cells, whole sections were used for DNA extractions, and samples with less than 70% tumor cells were sectioned and needle dissected to enrich for tumor cells. Genomic DNA from fresh-frozen and formalin-fixed paraffin embedded tissue was extracted using the DNeasy Blood & Tissue Kit (Qiagen) according to the manufacturer’s instructions. Purified DNA was quantified using the Qubit dsDNA BR Assay (Thermo Fisher Scientific).

## 1.2.2 Targeted DNA capture and sequencing

A custom panel of 32 genes (Supplementary Table S3) associated with ovarian cancer, homologous recombination and DNA repair was curated from the literature. Between 200 ng and 500 ng of input DNA was fragmented using an E220 ultrasonicator (Covaris). DNA libraries were constructed using the KAPA Hyper Prep Kit (Kapa Biosystems), and samples indexed using SeqCap adapter kits A or B (Roche). DNA libraries were pooled (8 samples per capture reaction for DNA extracted from fresh frozen tumor or blood, 3 to 4 samples per capture reaction for DNA extracted from formalin-fixed paraffin embedded tissue) and pooled samples were enriched for all exons and flanking intronic sequences of the target genes using xGen Predesigned Lockdown Probes (Integrated DNA Technologies). Captured samples were sequenced on the NextSeq platform (up to 24 samples per run) using the Mid-Output 75 bp paired-end protocol (Illumina). The mean coverage of target bases ranged from 124 to 985 per sample, with a median of 456-fold base coverage.

## 1.2.3 Targeted sequence alignment and variant detection

Targeted-sequencing data was processed and analyzed using an in-house bioinformatics pipeline constructed using Seqliner v0.1a (http://bioinformatics.petermac.org/seqliner): Raw reads (fastq files) were first quality checked using FastQC v0.11.2 (<http://www.bioinformatics.babraham.ac.uk/projects/fastqc/>) and trimmed using cutadapt v1.7.1 (3) to ensure high read qualities. Filtered reads were then aligned to the human reference genome (GRCh37/hg19) using BWA v0.7.12 (4) and duplicate reads marked using Picard (v1.119; <http://broadinstitute.github.io/picard>). INDEL realignment and base quality recalibration were performed using GATK v3.2.2 (5) prior to variant calling. GATK’s Unified Genotyper was used to call variants in the tumor samples. CONTRA v2.0.6 (6) was used to analyze copy variations based on the ratio of read coverage between the tumor and a germline control sample. Annotation of variants was performed using a local copy of the Ensembl (7) version R73 database and a customized version of Ensembl Variant Effect Predictor.

High-confidence variants were identified by filtering single nucleotide variants (SNVs) and indels to include those that were supported by: bidirectional reads, at least 8 reads, and an allele frequency of ≥ 5%. To identify pathogenic mutations, high-confidence variants were filtered to remove silent (synonymous) changes, as well as common variants with a global minor allele frequency (MAF) > 0.01 in either dbSNP (<https://www.ncbi.nlm.nih.gov/SNP/>) or the Exome Variant Server (EVS; <http://evs.gs.washington.edu/EVS/>), to exclude polymorphic sites.

## 1.2.4Mutation analysis

Mutations reported in this study were only those deemed pathogenic, that is nonsense, splice site and frameshift mutations that result in an early stop codon, and missense variants previously reported as pathogenic in published literature or curated databases, including the IARC (International Agency for Research on Cancer) TP53 Database (<http://p53.iarc.fr/>), ClinVar (<https://www.ncbi.nlm.nih.gov/clinvar/>), NHGRI Breast Cancer Information Core (<https://research.nhgri.nih.gov/bic/>), kConFab (http://www.kconfab.org) and the Human Gene Mutation Database (<http://www.hgmd.cf.ac.uk>). Missense point mutations previously found to be of low clinical significance, or those with unknown clinical significance, were excluded. Each mutation was annotated with respect to genomic variant and predicted transcript and protein changes according to the Human Genome Variation Society (HGVS) nomenclature (Supplementary Tables S4 and S5). A tumor was considered to have loss of heterozygosity if the mutant allele ratio was ≥ 0.7. Manual review was performed on all variants highlighted in this study using Integrative Genomics Viewer (8).

Large rearrangements that disrupt genes such as deletions and duplications of exons were considered pathogenic. These structural variants were only reported if supported by orthogonal evidence, such as detection by clinical sequencing in the paired germline sample, or by an orthogonal approach using SNP array data where available. Methods of detection and verification are listed in Supplementary Tables S4 and S5.

## 1.2.5 TCGA cohort

TCGA HGSC cohort genetic alterations were downloaded from cBioPortal (<http://www.cbioportal.org/>). Only tumors with exome sequence and copy number variant data were included (*n* = 316). The TCGA cohort was assessed for the proportion of cases affected by HR pathway inactivation, using the same genes in the targeted panel (Supplementary Table S3), and using consistent criteria for assigning pathogenicity. That is, loss-of-function (nonsense, splice site and frameshift) mutations that result in an early stop codon, and missense variants previously reported as pathogenic in published literature or curated databases (as listed in the previous section). Missense point mutations previously found to be of low clinical significance (benign), or those with unknown or uncertain clinical significance, were excluded.

TCGA tumor samples with homozygous deletion of *PTEN* were categorized as HR-deficient. We note that whole-gene homozygous deletion was only assessable in the subset of *Exceptional Responders* for which SNP array copy number data was available (*n* = 19), therefore the number of *Exceptional Responders* affected by whole-gene *PTEN* homozygous deletion (*n* = 1) may be an underestimate.

## 1.3 Promoter methylation analysis

## 1.3.1 Sodium bisulfite DNA modification

DNA (200 ng) was sodium bisulfite modified using the EZ DNA Methylation-Lightning™ Kit (Zymo Research, Irvine, CA) as per the manufacturer’s protocol. CpGenome Universal Methylated DNA (Millipore, Billerica, MA) was used as the fully methylated control DNA (100%). For each modification experiment, similar amounts of fully methylated DNA and DNA extracted from an unmethylated cell line (HCT116) were also modified.

## 1.3.2 *BRCA1* methylation analysis

The BRCA1 methylation-sensitive high resolution melting (MS-HRM) primers (forward: 5’-TtgTtgTttagcggtagTTTTttggtt; reverse: 5’-caAtcgcaAttttaatttatctAtaattccc) assessed 4 CpG dinucleotides (#37, #29, #21, #19) in a 79 bp amplicon (9). The forward primer was biotinylated to enable Pyrosequencing. The reverse primer was used as the sequencing primer. Bisulfite-modified fully methylated DNA was diluted in unmethylated bisulfite-modified DNA to obtain standards comprising 100%, 50%, 10%, 3%, and 0% methylated alleles. Each bisulfite-modified tumor DNA sample was run in duplicate on the CFX Connect™ Real-Time System (Bio-Rad, Hercules, US). Reactions were carried out in Hard-Shell® Low-Profile Thin-Wall 96-Well Skirted PCR Plates (Bio-Rad, Hercules, CA) in a 20 μl final reaction volume comprising 1X PCR Buffer (Qiagen), additional MgCl2 (Qiagen) to a final concentration of 2.5 mmol/L, 200 μmol/L of each deoxynucleotide triphosphate (Fisher Biotec, Perth, Australia), 200 nmol/L of the forward primer and 200 nmol/L of the reverse primer, 5 μmol/L of SYTO 9 (Invitrogen, Carlsbad, CA), 0.5 units of HotStar Taq DNA polymerase (Qiagen) and 2 μl of Bisulfite-modified DNA. The PCR conditions included an initial denaturation of 15 minutes at 95°C, followed by 50 cycles of 10 seconds at 95°C, 10 seconds at 60°C, 20 seconds at 72°C; 1 cycle of 1 minute at 97°C and a high-resolution melting (HRM) step from 70°C to 90°C, increasing at 0.2°C per second. The methylation level of each DNA sample was determined visually by comparing it against the melting profiles of the standards.

## 1.3.3 *RAD51C* methylation analysis

The RAD51C methylation-sensitive high resolution melting (MS-HRM) primers (forward: 5’- GAAAATTTAtAAGAtTGCGtAAAGtTGtAAGG; reverse: 5’- CTAACCCCGaAaCAaCCAAACTCC) amplified a 142 bp amplicon. Bisulfite-modified fully methylated DNA was diluted in unmethylated bisulfite-modified DNA to obtain standards comprising 100%, 50%, 10%, 3%, and 0% methylated alleles. Each bisulfite-modified tumor DNA samples was run in duplicate on the CFX Connect™ Real-Time System (Bio-Rad, Hercules, CA). Reactions were carried out in Hard-Shell® Low-Profile Thin-Wall 96-Well Skirted PCR Plates (Bio-Rad, Hercules, US) in a 20 μl final reaction volume comprising 1X PCR Buffer (Qiagen) containing 1.5 mmol/L of MgCl2, 200 μmol/L of each deoxynucleotide triphosphate (Fisher Biotec, Perth, Australia), 200 nmol/L of the forward primer and 200 nmol/L of the reverse primer, 5 μmol/L of SYTO 9 (Invitrogen, Carlsbad, CA), 0.5 units of HotStar Taq DNA polymerase (Qiagen) and 2 μl of Bisulfite-modified DNA. The PCR conditions included an initial denaturation of 15 minutes at 95°C, followed by 50 cycles of 10 seconds at 95°C, 20 seconds at 64°C, 30 seconds at 72°C; 1 cycle of 1 minute at 97°C and a high-resolution melting (HRM) step from 75°C to 90°C, increasing at 0.2°C per second. The methylation level of each DNA sample was determined visually by comparing it against the melting profiles of the standards.

## 1.3.4 Bisulfite pyrosequencing of amplified products

Selected amplified products were bisulfite pyrosequenced using the Qseq Pyrosequencer (Bio Molecular Systems, Upper Coomera, Australia). The PyroMark Gold Q24 Reagents (Qiagen) were used and the reactions were carried out using in 48 Well Discs (Gene Target Solutions, Dural, Australia) in a 13 μl final reaction volume, comprising 1μl Streptavidin Mag Sepharose (GE Healthcare, Little Chalfont, UK), 5 μl of PCR product and 2 μl of the reverse primer (5 mmol/L).

# 2. SUPPLEMENTARY FIGURES

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Supplementary Figure S1. **Outline of cohort selection and analyses.**

Cases included in the study were from the Australian Ovarian Cancer Study (AOCS) and the Gynaecological Oncology Biobank (GynBiobank). Advanced stage (FIGO IIIC/IV), serous ovarian cancer cases (including primary peritoneal and fallopian tube cancer) meeting clinical, pathological and surgical criteria for *Exceptional Responders* were selected. Comparison control cohorts were identified the same patient populations as the *Exceptional Responders*, matched for stage and histology and included all cases, except those that met the exceptional outcome criteria. The numbers of cases in each group and specific analysis are indicated. EOC, epithelial ovarian cancer; PFS, progression-free survival; OS, overall survival; LGSC, low-grade serous cancer; HGSC, high-grade serous cancer; RD, residual disease; TMA, tissue microarray; *Long-PFS*, Long-Progression-Free Survival; *MR*, Multiple Responders; *LTS*, Long-Term Survivors; TE, tumor epithelium; STR, stromal; g*BRCA1/2*, germline *BRCA1/2*;HR pathway status, homologous recombination pathway gene alteration status. *References*: a Cancer Genome Atlas Research Network, Nature 474:609-15, 2011; b Alsop *et al*, J Clin Oncol 30:2654-63, 2012; c Gyorffy *et al,* Endocr Relat Cancer 19:197-208, 2012

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy.**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy (cont’d).**

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Supplementary Figure S2. **Clinical response and therapy course of 96 patients with exceptional responses to chemotherapy.**

Plots of tumor marker cancer antigen 125 (CA125) levels over time from all patients classified as *Exceptional Responders*. Plots are grouped by chemosensitivity categories, including *Long-Term Survivors* that are progression-free, *Long-Term Survivors* with progression, *Multiple Responders*, and *Long-PFS* patients with > 1 cm residual disease and ≤ 1 cm residual disease. Black lines represent CA125 levels on a log scale, and dotted grey lines represent the upper limit of normal for each CA125 measurement. The upper limit of normal can vary depending on the type of CA125 assay performed. Colored circles and rectangles represent administration of different lines of treatment as indicated. Also indicated is the time of primary surgery (blue triangle), first progression (red triangle), and death (grey cross) or date last seen alive (green diamond).

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Supplementary Figure S3. **Distribution and type of *TP53* mutations.**

**A,** Lollypop symbols indicate the amino acid (aa) location (*x*-axis) of missense (green), in-frame (brown) and truncating (black) mutations in all *Exceptional Responders* with a *TP53* mutation (*n* = 80). The height of the lollypop (*y*-axis) represents the number of mutations identified at the given residue. Colored rectangles indicate functional domains of the p53 protein: green, transactivation motif; red, DNA-binding domain; blue, tetramerization motif. Lollypop figure was generated using MutationMapper software v1.0.1 (cBioPortal). **B,** Proportions of missense and truncating (nonsense, frameshift and splice site) mutations in each patient subgroup, compared to an independent study (10) (The Cancer Genome Atlas, TCGA; Chi-square *P* values reported). **C,** Kaplan-Meier estimates of progression-free survival and overall survival for 265 HGSC patients with *TP53* mutations in TCGA, according to *TP53* mutation category. ER, Exceptional Responder.

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Supplementary Figure S4. **RB1 protein expression altered by genomic inactivation.**

Association between genomic *RB1* inactivation (homozygous deletion or gene breakage detected by whole-genome sequencing (1)) and RB1 protein expression (detected by immunohistochemistry) in HGSC tumors (*n* = 33, Chi-square *P* value reported).

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Supplementary Figure S5. **Characterization of CD8 and Ki-67 in tumors according to homologous recombination mutation status.**

**A,** The average density (in cells/mm2) of CD8+ lymphocytes in both tumor epithelium (TE) and **B,** stroma (STR). **C,** Quantification of Ki-67 expression in tumor nuclei, as detected by immunohistochemical staining. Horizontal lines indicate median scores. The *Exceptional Responder* cohort is broken down into molecular subgroups. *P* values are shown for the subgroups that had significant differences to the median score of unselected HGSC (Mann Whitney test).

# 3. SUPPLEMENTARY TABLES

Supplementary Table S1

Histotype classification

Supplementary Table S2

Immunohistochemical analysis: primary antibodies and staining conditions

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Supplementary Table S3

Homologous recombination and DNA repair panel

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Supplementary Table S4

*TP53* mutations

Supplementary Table S5

Loss-of-function mutations in homologous recombination pathway genes

Supplementary Table S6

Comparison of molecular alteration prevalence between clinical subgroups

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Supplementary Table S7

Patient characteristics of tissue microarray cohort

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