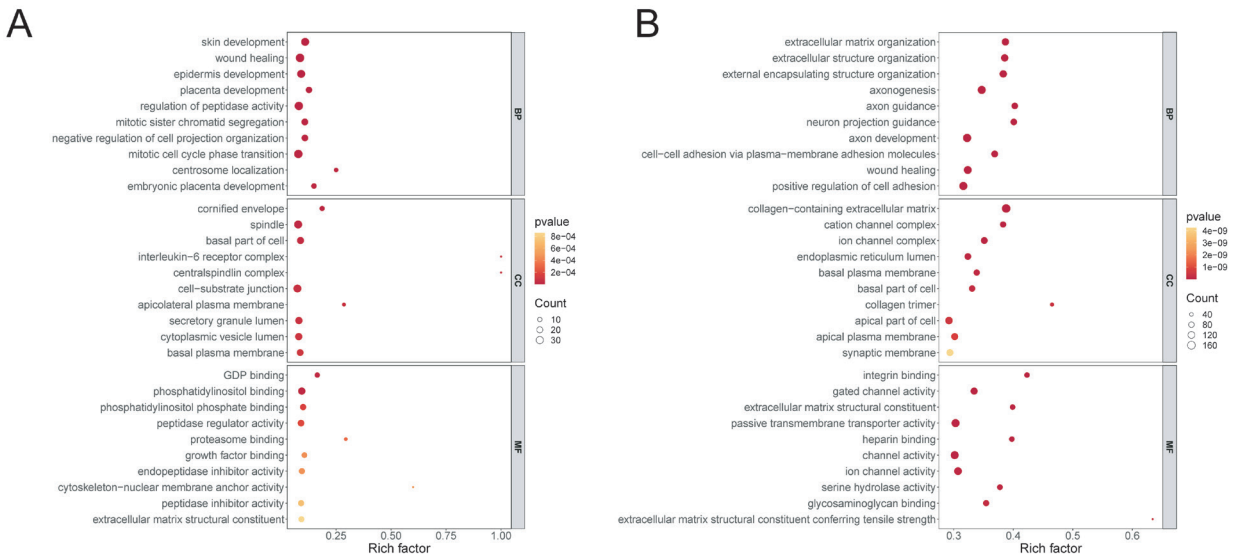

Figure S1 Functional enrichment analysis of differentially expressed genes (DEGs) between HCC827 and HCC827/ER cells. (A) Gene Ontology enrichment of up-regulated DEGs in HCC827/Er cells. (B) GO enrichment of down-regulated DEGs in HCC827/ER cells.

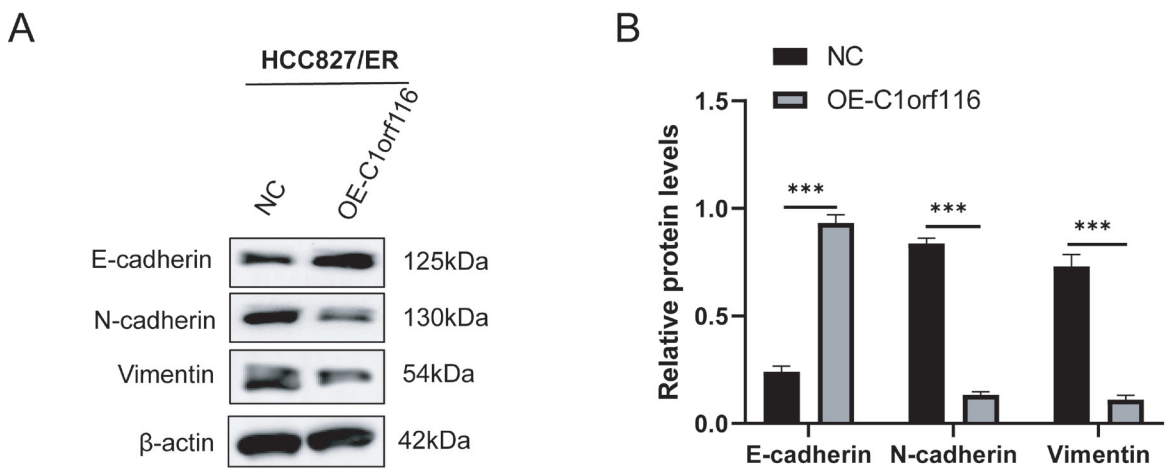

Figure S2 Over-expression of C1orf116 inhibited EMT in HCC827/ER. (A) Western blotting was performed to analyze the expression of EMT markers (E-cadherin, N-cadherin, and vimentin) in HCC827/ER cells. (B) Relative protein levels of E-cadherin, N-cadherin, and vimentin were quantified in HCC827/ER cells. ***, P<0.001.

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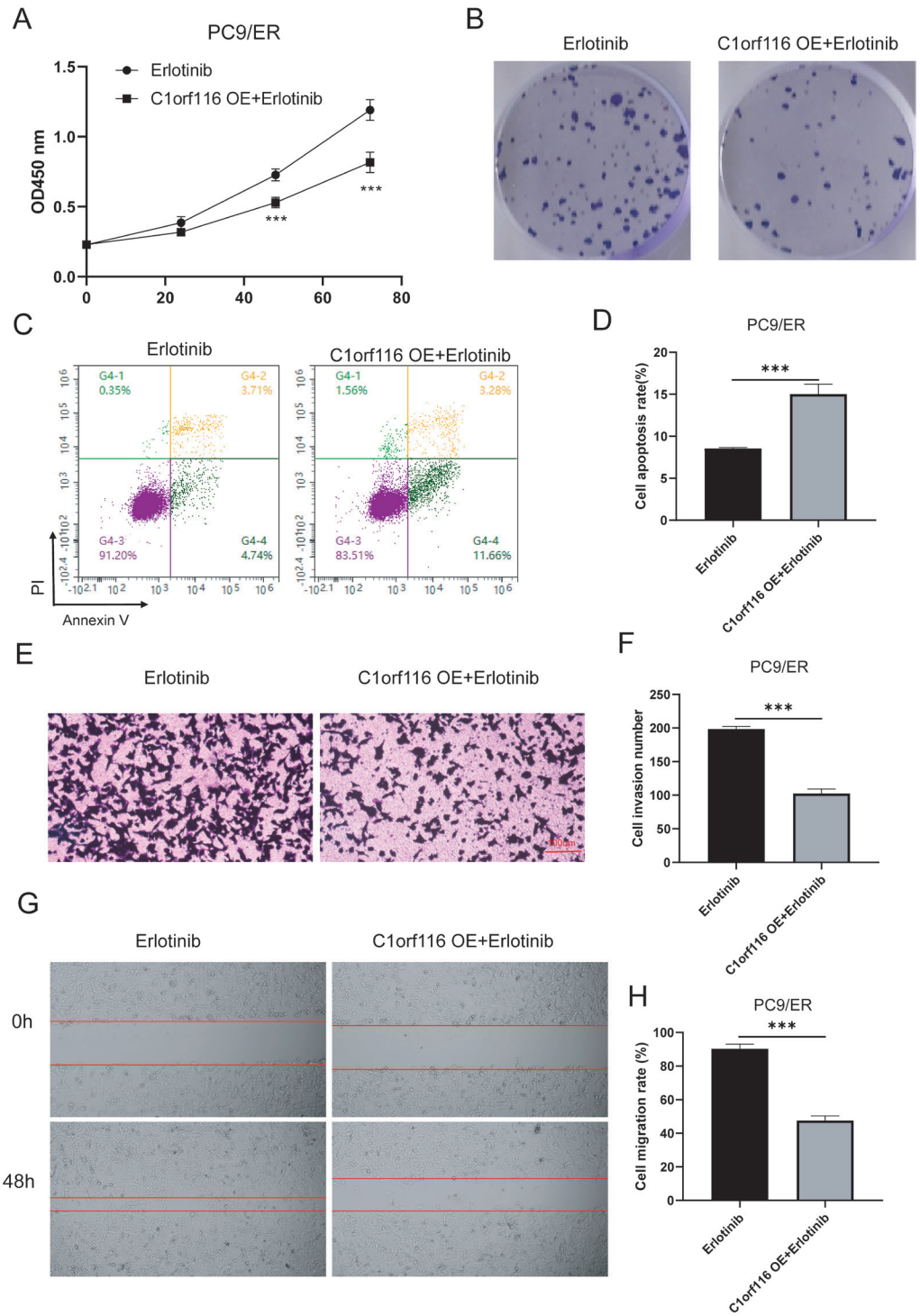

Figure S3 C1orf116 increased erlotinib sensitivity in PC9/ER cells. (A) Effect of C1orf116 on erlotinib efficacy was detected by CCK-8 assay. (B) The ant-proliferative effect of C1orf116 was examined using the colony formation assay in combination with indicated erlotinib in PC9/ER cells. (C) Cell apoptosis was analyzed by FACS. (D) Cell apoptosis rate was quantified. (E) The cell invasion was detected by Transwell assay in PC9/ER. (F) Cell invasion number was quantified. (G) Area wound healed was used to detect cell migration in PC9/ER. (H) Cell migration rate was quantified. Data are presented mean ± SD from five randomly selected visual fields. ***, P<0.001.

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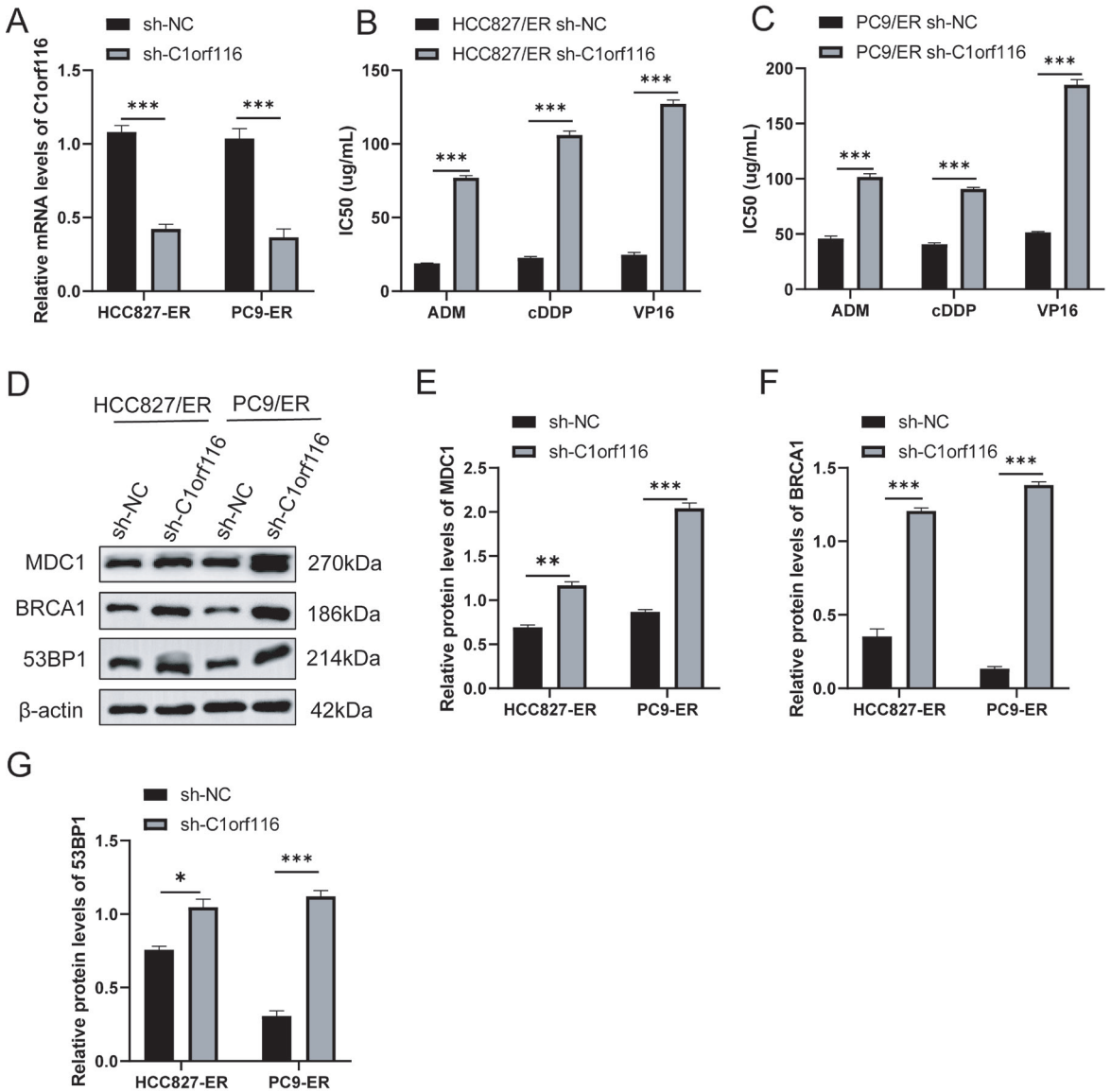

Figure S4 C1orf116 knockdown promotes multidrug resistance (MDR) and upregulates DNA damage repair proteins in erlotinib-resistant LUAD cells. (A) qRT-PCR validation of C1orf116 knockdown efficiency in both cell lines. IC50 values for ADM, cDDP, and VP16 in HCC827/ER (B) and PC9/ER (C) cells after C1orf116 knockdown. Data represent mean ± SD from three independent experiments (***P<0.001, **P<0.01, unpaired *t*-test). (D-G) Western blot analysis of DDR proteins (BRCA1, MDC1, 53BP1) in HCC827/ER and PC9/ER cells transfected with sh-NC or sh-C1orf116. β-actin served as the loading control. Bar graphs show relative protein levels normalized to β-actin (mean ± SD, n=3).

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