Figure S1. Expression value of LAPTM5 in primary or spinal metastatic ER$^+$ BC. LAPTM5 expression was significantly downregulated in SM tissues compared with that in the primary ER$^+$ BC tissue. Data (GSE14661) was obtained from GEO datasets. LAPTM5, lysosomal protein transmembrane 5; ER$^+$ BC, estrogen receptor-positive breast cancer; SM, spinal metastasis.

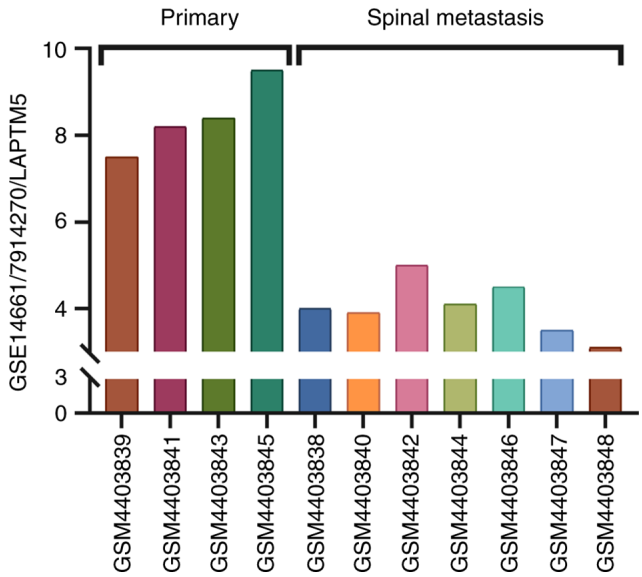

Figure S2. LAPTM5 is negatively related to the proliferation and migration *in vitro* and tumorigenesis *in vivo* of ER$^+$ BC cells under docetaxel treatment. (A and B) Quantitative analysis of the protein levels of LAPTM5 in the MCF-7 and T47D cell lines in Fig. 2A. (C) Colony formation results of blank, LAPTM5-sh3 (knockdown) and LAPTM5-OE (overexpressing) T47D cells with or without docetaxel treatment. (D) Statistical analysis of the colony formation assay. ***P<0.001, compared to the blank T47D cells. (E) Wound healing results of blank, LAPTM5-sh3 and LAPTM5-OE T47D cells with or without docetaxel treatment. (F) Statistical analysis of the wound healing assay. ***P<0.001, compared to the blank T47D cells. (G and H) Statistical analysis of Ki-67 and TUNEL staining in Fig. 2K. **P<0.01, and ***P<0.001, compared to the bland MCF-7 cells. LAPTM5, lysosomal protein transmembrane 5; ER$^+$ BC, estrogen receptor-positive breast cancer.

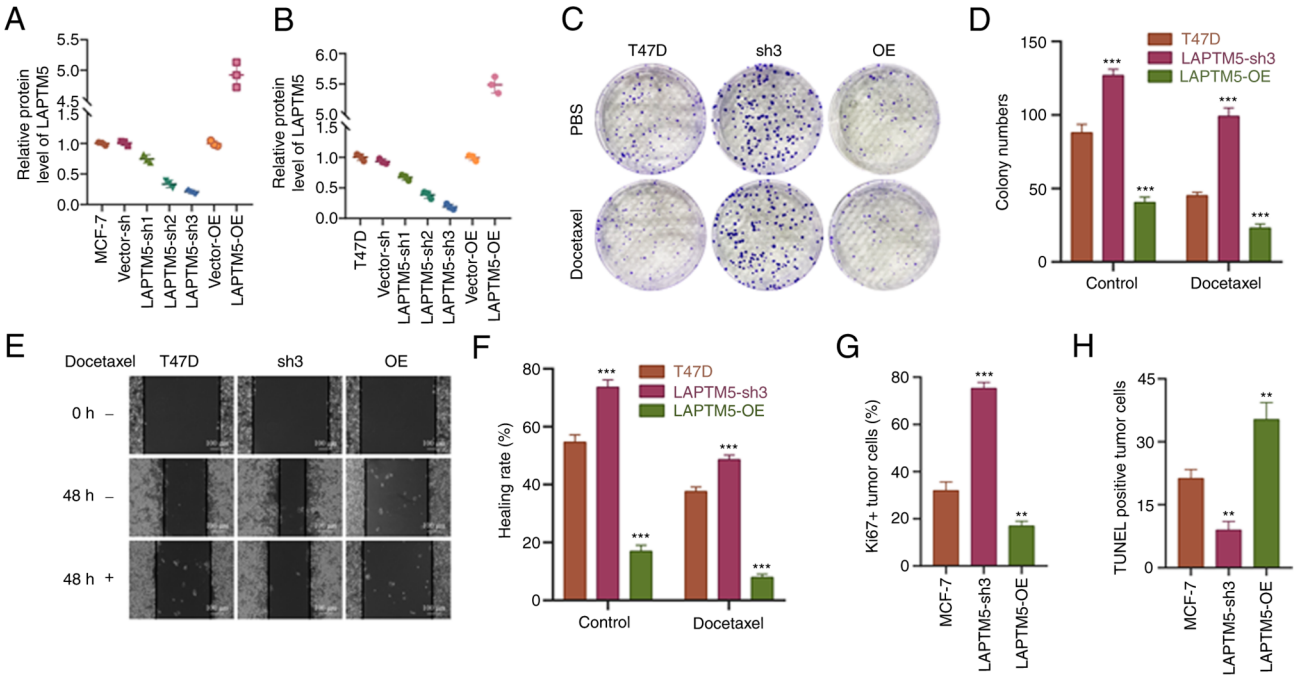

Figure S3. Gene set enrichment analysis (GSEA) of data from GEO dataset (GSE14661). The color tints indicate the P-values. The size of the circle presented the number of selected genes in the pathway. Forty genes were found to be enriched in carbon metabolism, indicated that LAPTM5 expression is strongly correlated with the metabolism of ER^+ BC. LAPTM5, lysosomal protein transmembrane 5; ER^+ BC, estrogen receptor-positive breast cancer.

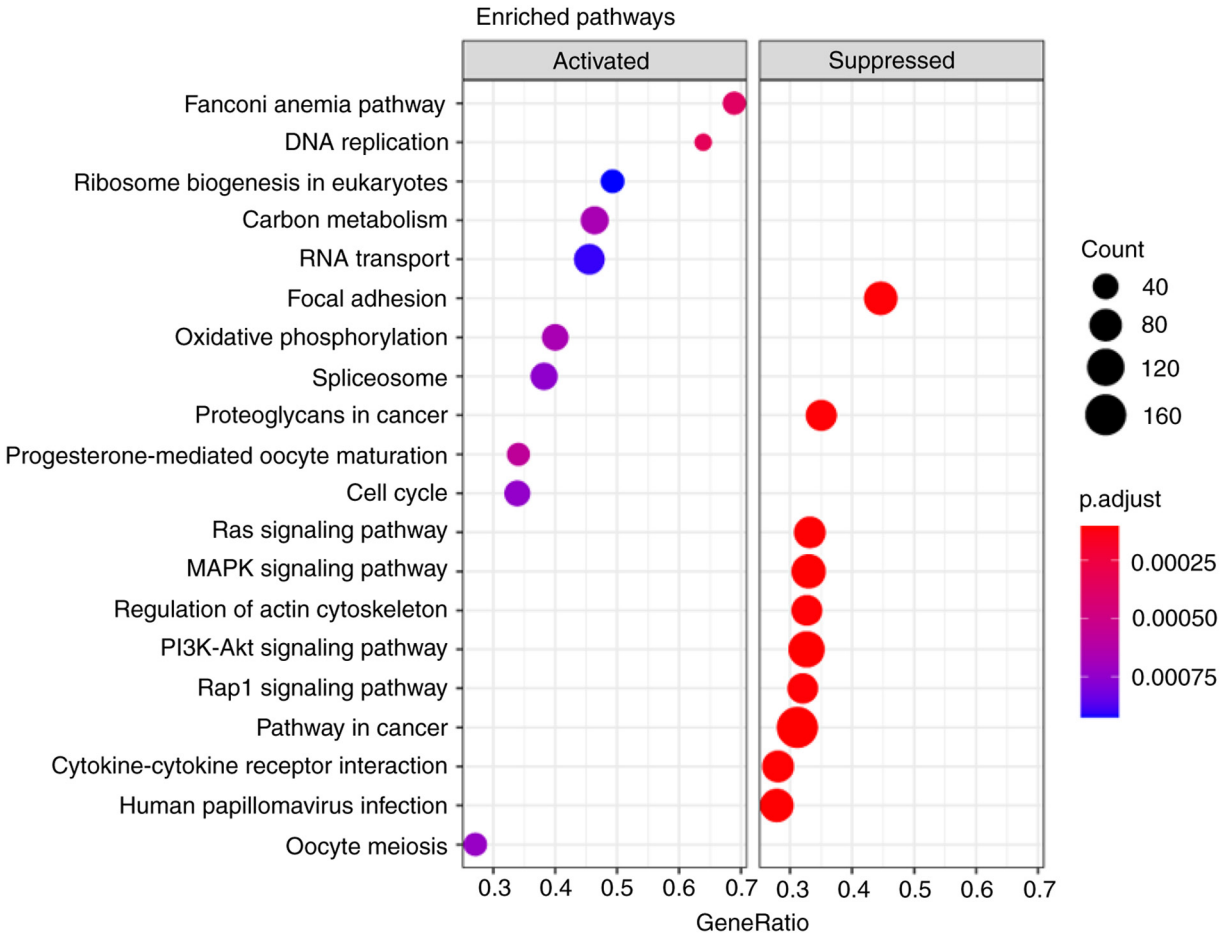

Figure S4. Expression of GLS2 between LAPTM5-silenced (sh3) and control ER$^+$ BC cells. LAPTM5, lysosomal protein transmembrane 5; ER$^+$ BC, estrogen receptor-positive breast cancer; GLS2, glutaminase 2.

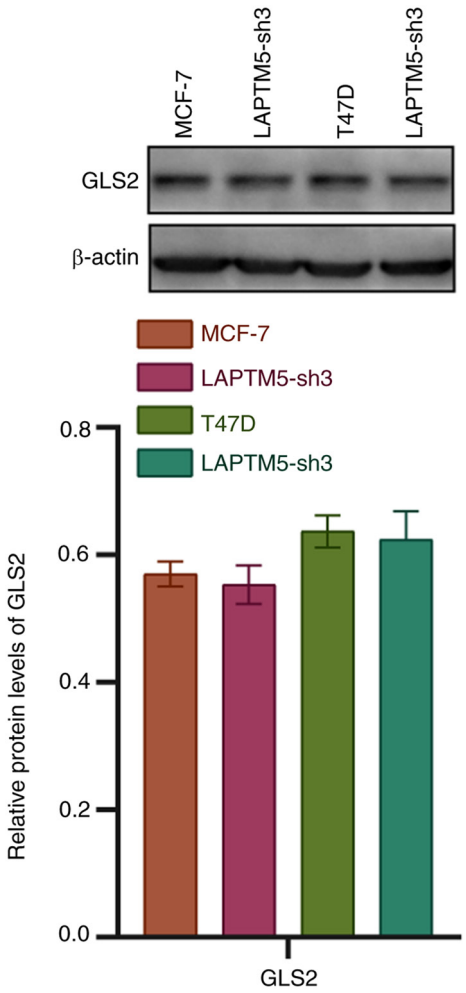

Figure S5. Blockade of CX3CL1/CX3CR1 and glutaminase inhibits SM and enhances the chemosensitivity of ER$^+$ BC cells. (A) Image showing SM of ER$^+$ BC *ex vivo*. (B) Kaplan-Meier spinal metastasis-free curve of the mouse groups. (C) Kaplan-Meier survival curve. Blank MCF-7 cells were used as the control group (a). The other groups were established with LAPTM5-sh3 cells, and groups b-f were injected with saline (b), docetaxel (c), BPTES + docetaxel (d), JMS-17-2+docetaxel (e), and BPTES + JMS-17-2+docetaxel (f). LAPTM5, lysosomal protein transmembrane 5; ER+ BC, estrogen receptor-positive breast cancer; CX3CL1, C-X3-C motif chemokine ligand 1; CX3CR1, C-X3-C motif chemokine receptor 1; SM, spinal metastasis.

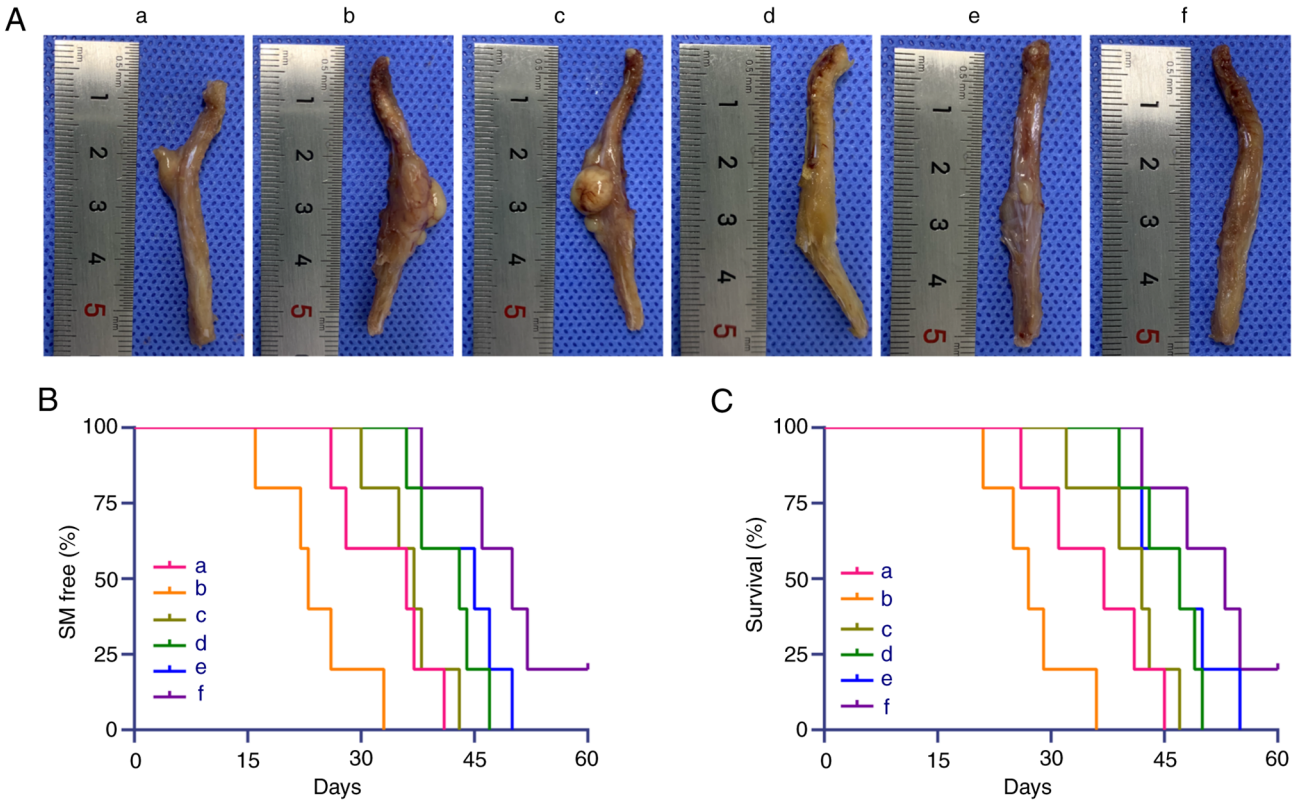

Table SI. Patient clinicopathological data.

Patient no.	Age (years)	Tumor type	Pathological results
Patient 1	52	Primary ER^+ BC	ER++ (70%)
Patient 2	60	Primary ER^+ BC	ER+++
Patient 3	45	Primary ER^+ BC	ER+++ (90%)
Patient 4	48	Primary ER^+ BC	ER+++ (90%)
Patient 5	51	Primary ER^+ BC	ER++
Patient 6	48	Primary ER^+ BC	ER+++ (90%)
Patient 7	53	SM of ER^+ BC	ER++ (90%)
Patient 8	43	SM of ER^+ BC	ER+ (80%)
Patient 9	53	SM of ER^+ BC	ER+++ (100%)
Patient 10	49	SM of ER^+ BC	ER+ (100%)
Patient 11	56	SM of ER^+ BC	ER++ (80%)
Patient 12	55	SM of ER^+ BC	ER+++ (90%)
Patient 13	60	SM of ER^+ BC	ER+++ (100%)

ER^+, estrogen receptor-positive; BC, breast cancer; SM, spinal metastasis.