Methods:

1. SUPPLEMENTARY TABLES

Table S1. Primer information of qRT-PCR

Table 1 Primer information of qRT-PCR

Gene names	Primer names	Sequence	Length
GAPDH	GAPDH-F	TGGTATCGTGGAAGGACTC	19
	GAPDH-R	GGATGATGTTCTGGAGAGC	19
PERK	PERK-F	TCTTGGTCCCACTGGAAGAG	20
	PERK-R	AAAGCAGTGGGATTTGGATG	20
ATF3	ATF3-F	GTGCCGAAACAAGAAGAAGG	20
	ATF-R	TCTGAGCCTTCAGTTCAGCA	20
ATF4	ATF4-F	GTGTTCTCTGTGGGTCTGCC	20
	ATF4-R	GAGCCTCGTTCTTCTTTCC	20
DDIT3	DDIT3-F	GACCCTGCTTCTCTGGCTTG	20
	DDIT3-R	CCGTTTCCTGGTTCTCCCTT	20
BAX	BAX-F	TCTGACGGCAACTTCAACTG	20
	BAX-R	GGGACATCAGTCGCTTCAGT	20
BCL2	BCL-2-F	GTGTGTGGAGAGCGTCAACC	20
	BCL2-R	AGCCAGGAGAAATCAAACAG	20

Results: 2 SUPPLEMENTARY FIGURES Figure S1

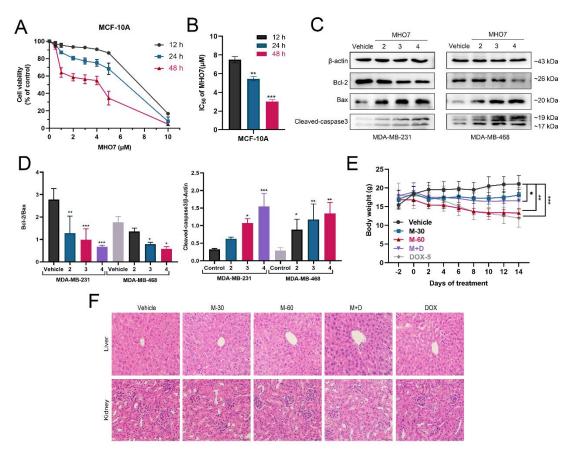


Figure S1. Antitumor effect of MHO7 in vitro and in vivo. (A) The effect of MHO7 on MCF-10A cells at 12, 24, and 48 h. (B) The IC₅₀ value of MHO7 on MCF-10A at 12, 24, and 48 h. (C-D) The expression of Bxl2-2, Bax and cleaved caspase 3 were measured by western blot after the treatment of MHO7. (E) Body weight-time curves were measured in different groups. (F) Liver and kidney tissue in mice were analyzed H&E staining.

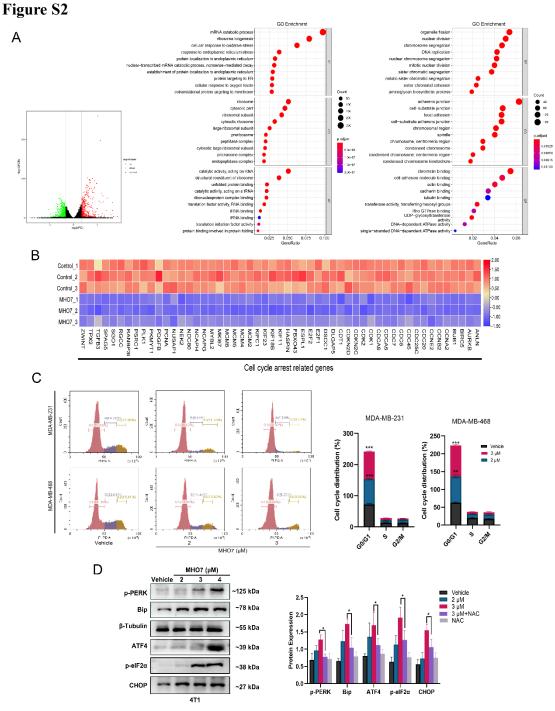


Figure S2. RNA-seq analysis revealed the induction of ER stress and cell cycle arrest in MHO7 treated MDA-MB-231 cells. (A) Volcano Plot and GO enrichment of DEGs were analyzed by RNA-seq. (B) Heat map of differentially expressed cell cycle-related genes was analyzed by RNA-seq. (C) Cell cycle was detected by flow cytometry after MHO7 treatment. (D) The expression of BiP/p-PERK/p-eIF 2α /ATF4/CHOP were measured by western blot under MHO7 treatment in 4T1 cells.

Figure S3

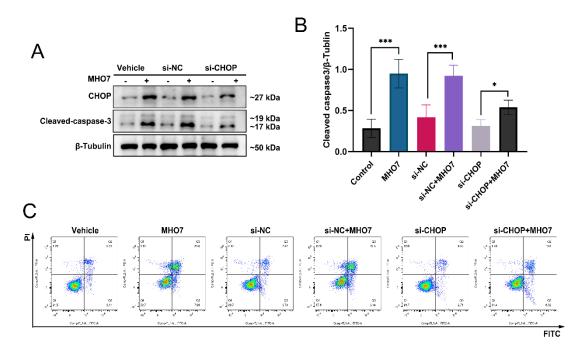


Figure S3. CHOP contributed to the apoptosis induced by MHO7. (A-B) CHOP and caspase3 expression measured by western blot in MDA-MB-231 cells after MHO7 (3 μ M) or si-CHOP treatment. (C) The percentages of apoptosis cells were measured by flow cytometry under MHO7 (3 μ M) or si-CHOP treatment. *P < 0.05; **P < 0.01; ***P < 0.001; and **** P < 0.0001.

Figure S4

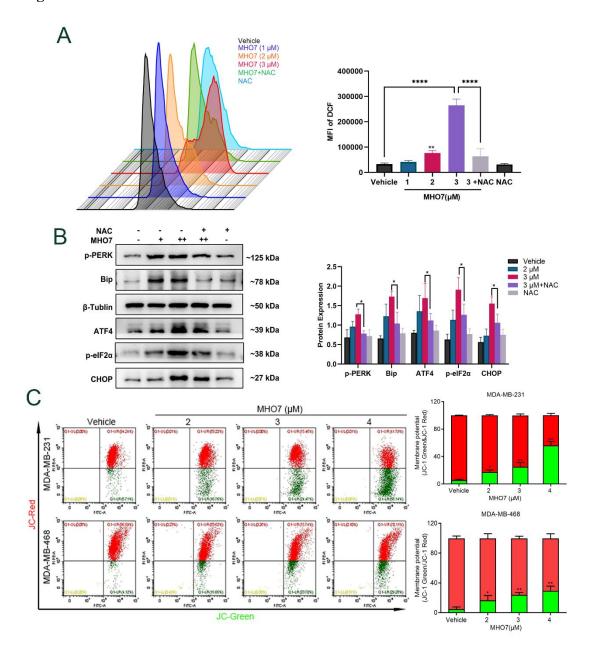


Figure S4. ROS generation and mitochondrial dysfunction induced by MHO7. (A) The ROS level was detected by flow cytometry when treated with MHO7 for 24 h or pretreated with NAC (4 mM) for 1 h in 4T1 cells. (B) ER stress-related proteins were measured by western blot under the treatment of MHO7 (+: 2 μ M; ++:3 μ M) or pretreated with NAC (4 mM) in 4T1 cells. (C) Mitochondrial membrane potentials (MMPs) were detected by flow cytometry after the treatment of MHO7. *P < 0.05; **P < 0.01;

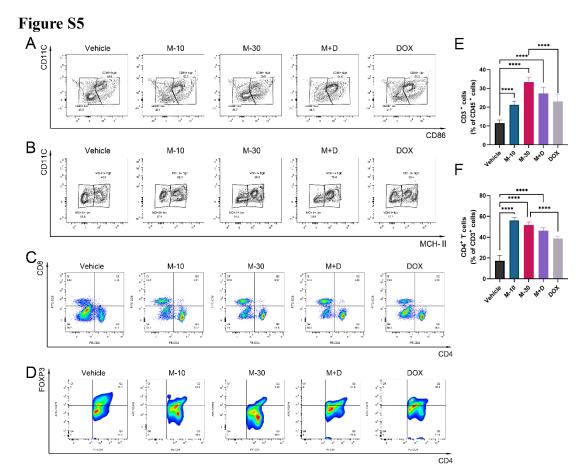


Figure S5. Maturation status of dendritic cells, activation of T cells and level of Tregs in MHO7 treated mice were analyzed by flow cytometry, respectively. (A) CD11C+/CD86+ DC cells in spleens were analyzed by flow cytometry. (B) CD11C+/MHC II DC cells in spleens were analyzed by flow cytometry. (C) CD3+/CD8+ T cells in spleens were analyzed by flow cytometry. (D) CD4+/FOXP3+ T cells in spleens were analyzed by flow cytometry. (E) CD3+/CD8+ T cells in spleens were analyzed by flow cytometry. (F) CD3+/CD4+ T cells were analyzed by flow cytometry. in spleens. *P < 0.05; **P < 0.01; ***P < 0.001; and ****P < 0.0001.

Figure S6

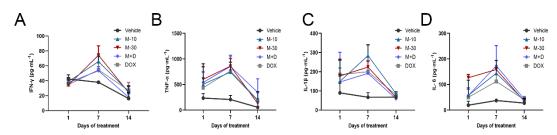


Figure S6. Antitumor cytokine levels, including (A) IFN- γ , (B) TNF- α , (C) IL-1 β and (D) IL-6 in the serum were detected by ELISA from each group on day 1,7,14 after treatment. (n=3).