Supporting Information

H_2O_2 induces nuclear transport of the receptor tyrosine kinase c-MET in breast cancer cells via a membrane-bounded retrograde trafficking mechanism

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Supporting information includes the following:

Figure S1

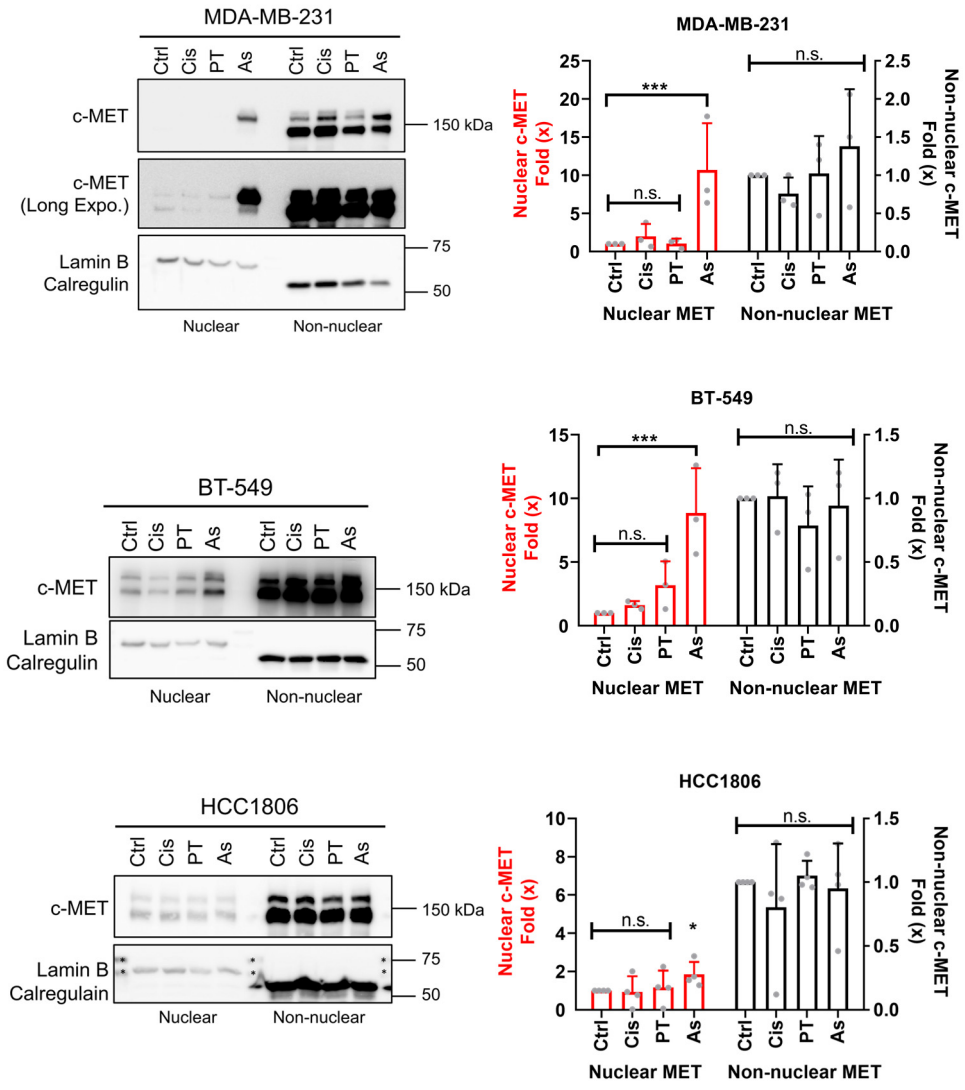

Fig S1. Nuclear accumulation of full-length c-MET in breast cancer cells after different chemotherapy drug treatments. Cells were treated overnight with either DMSO (Ctrl), 10 μM cisplatin (Cis), 1 μM paclitaxel (PT), or 10 μM sodium arsenite (As) overnight before harvested and subjected to cellular fractionation. Lamin B and calregulin were used as markers for nuclear and non-nuclear fractions. Fold change (x) of three independent experiments for MDA-MB-231 and BT-549 are indicated in histogram with mean ± S.D. Individual values are shown in dots. Fold change (x) of four independent HCC1806 experiments are indicated in histogram with mean ± S.D.

Figure S2.

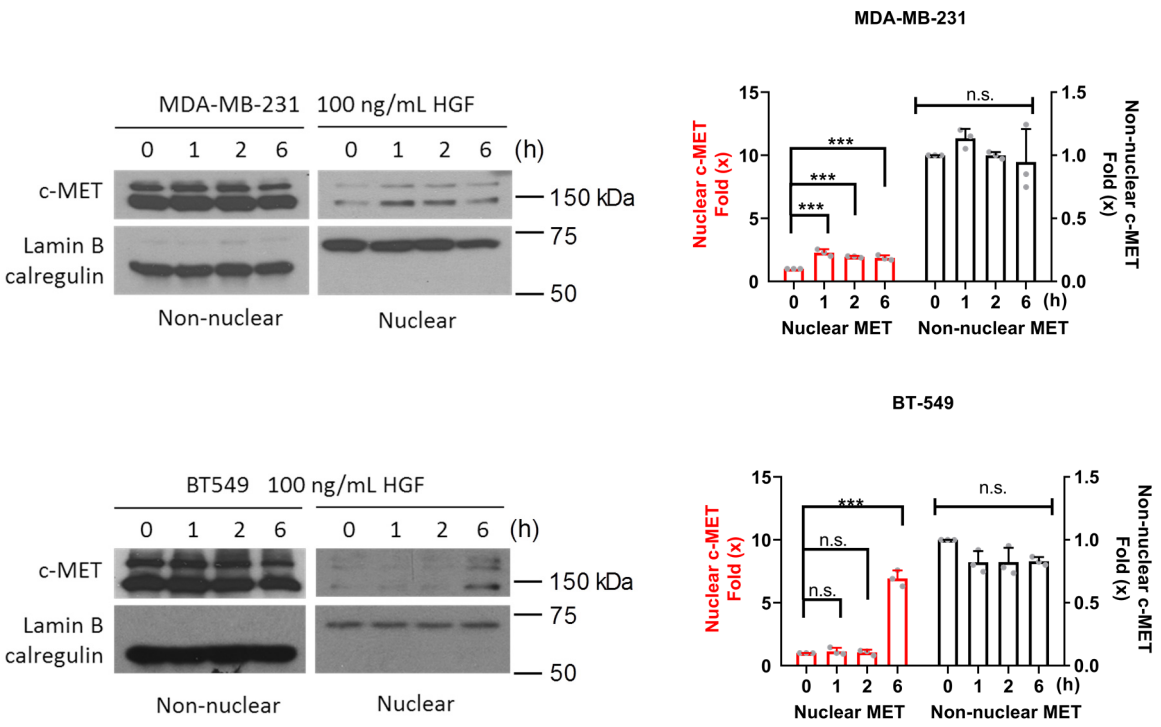

Fig. S2 Prolonged HGF stimulate full-length c-MET nuclear accumulation in breast cancer cells. MDA-MB-231 and BT-549 cells were treated with 100 ng/ml HGF for times indicated. Lamin B and calregulin were used as markers for nuclear and non-nuclear fractions. Statistical analysis of prolonged treatment and full-length c-MET nuclear accumulations in both cell lines. Fold change (x) of three independent experiments are indicated in histogram with mean ± S.D.