**Supplementary Figure 1. Chemotherapy induces multiple cell death-associated pathways.** 4T1 cells were treated with vehicle (media), 10ug/ml mafosfamide (MAF) or 1000ng/ml gemcitabine (GEM) for either (A) 4 hours or (B-F) 24 hours. Western blotting (representative of 2 independent blots) and flow cytometry (*n* = 3 per group over 2 independent experiments) were used to characterize cell death pathways activated by MAF or GEM treatment. Actin was used as a loading control in all western blots. A) Endoplasmic reticulum stress upon treatment with chemotherapy was assessed through phosphorylation of PERK (PKR-like endoplasmic reticulum kinase) and eIF2α (eukaryotic initiation factor). B) The cleavage of caspase-3 and C) annexin V staining were analyzed in 4T1 cells to assess apoptosis in response to MAF or GEM. \* *P* < 0.05 compared to vehicle. D) Lipidation of LC3-I (Microtubule-associated protein 1A/1B-light chain 3) to LC3-II and degradation of SQSTM-1 (Sequestrome-1; p62) are associated with autophagy. E) Phosphorylation of MLKL (mixed lineage kinase domain-like) is associated with necroptosis. F) The cleavage of caspase 1 and 7 are associated with pyroptosis.

**Supplementary Figure 2. NKT-cell activation following therapy.** Mice were inoculated with 2x105 4T1 mammary carcinoma cells and treated with gemcitabine (GEM) (30 mg/kg on day 12, 14, 16) or perioral cyclophosphamide (CPX) (20 mg/kg/day) from day 12-16. Splenocytes were harvested on day 17 to assess NKT cell activation status. As positive controls, some mice were treated with α-GalCer-loaded DCs 3 days prior to harvest or 3 hours prior to harvest (since IFNγ levels return to baseline by 72 hours following α-GalCer-loaded DC treatment). A) The number of NKT cells (TCRβ+CD1d tetramer+) and frequency of B) CD69+ NKT cells (expression is lost following NKT-cell activation) (4), C) IFNγ+ NKT cells, D) and Granzyme-B+ NKT cells were assessed by flow cytometry (*n* = 5 per group over 2 independent experiments). \* *P* < 0.05 compared to unloaded DCs.