

## **Breast Cancer Research & Treatment**

**Supplementary File:** HO-1 drives autophagy as a mechanism of resistance against HER2-targeted therapies

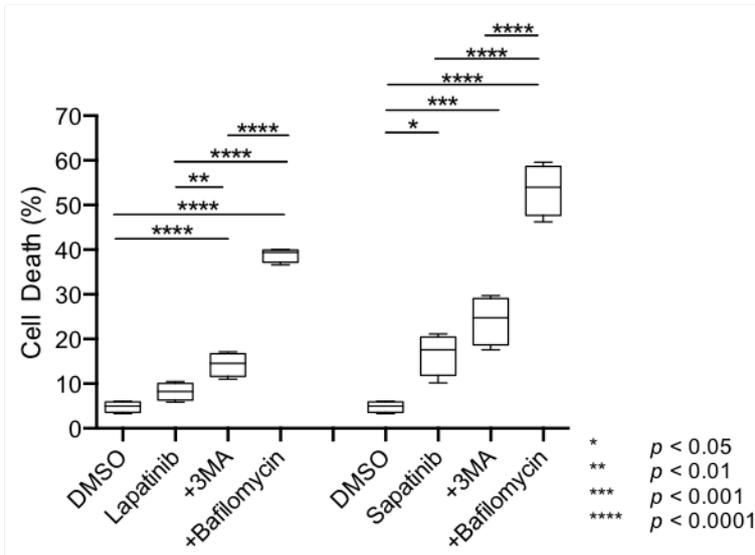
Natasha Tracey<sup>1</sup>, Helen Creedon<sup>1</sup>, Alain J Kemp<sup>1</sup>, Jayne Culley<sup>1</sup>, Morwenna Muir<sup>1</sup>, Teresa Klinowska<sup>2</sup>, Valerie G Brunton<sup>1\*</sup>

<sup>1</sup>Edinburgh Cancer Research UK Centre, Institute of Genetics & Molecular Medicine, University of Edinburgh, Crewe Road South, Edinburgh, EH4 2XR and <sup>2</sup>AstraZeneca, Oncology, IMED Biotech Unit, Cambridge, UK

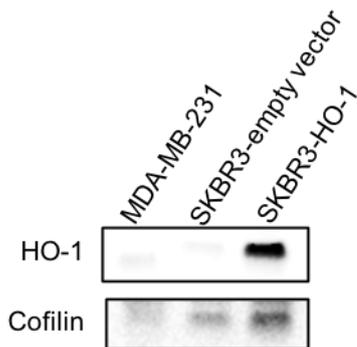
\*Correspondence: Professor Valerie Brunton  
Email [v.brunton@ed.ac.uk](mailto:v.brunton@ed.ac.uk)  
Phone +44 131 650 8500  
FAX +44 131 777 3520

## Supplementary Figure 1

a



b



**Supplementary Figure 1** MDA-MB-231 cells were treated for 48 hours with dimethyl sulfoxide (DMSO; 0.01%), sapatinib (0.67  $\mu\text{M}$ ) or lapatinib (5  $\mu\text{M}$ ) in the presence or absence of autophagy inhibitors 3-methyladenine (3-MA; 5 mM) or bafilomycin A1 (bafilomycin; 5 nM). Cells were stained with propidium iodide and percentage of cell death was analysed using an Accuri™ C6 Flow Cytometer. Results presented as box and whisker plot, minimum of three biological repeats. All conditions were compared to DMSO control and single agent treatments. One-way ANOVA, Bonferroni's post-hoc test, not significant=NS,  $p < 0.05 = *$ ,  $p < 0.01 = **$ ,  $p < 0.001 = ***$ . (b) Western blot analysis of HO-1 in SKBR3 vector, SKBR3-HO-1 and MDA-MB-231 cells. Cofilin was used as a loading control