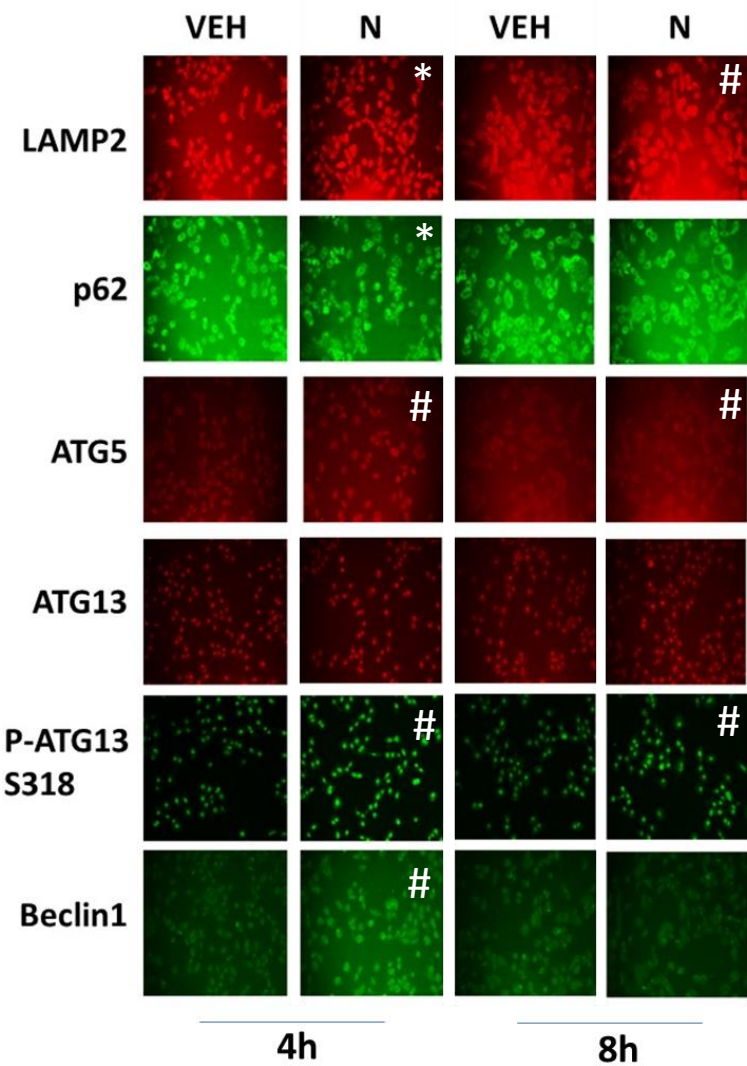
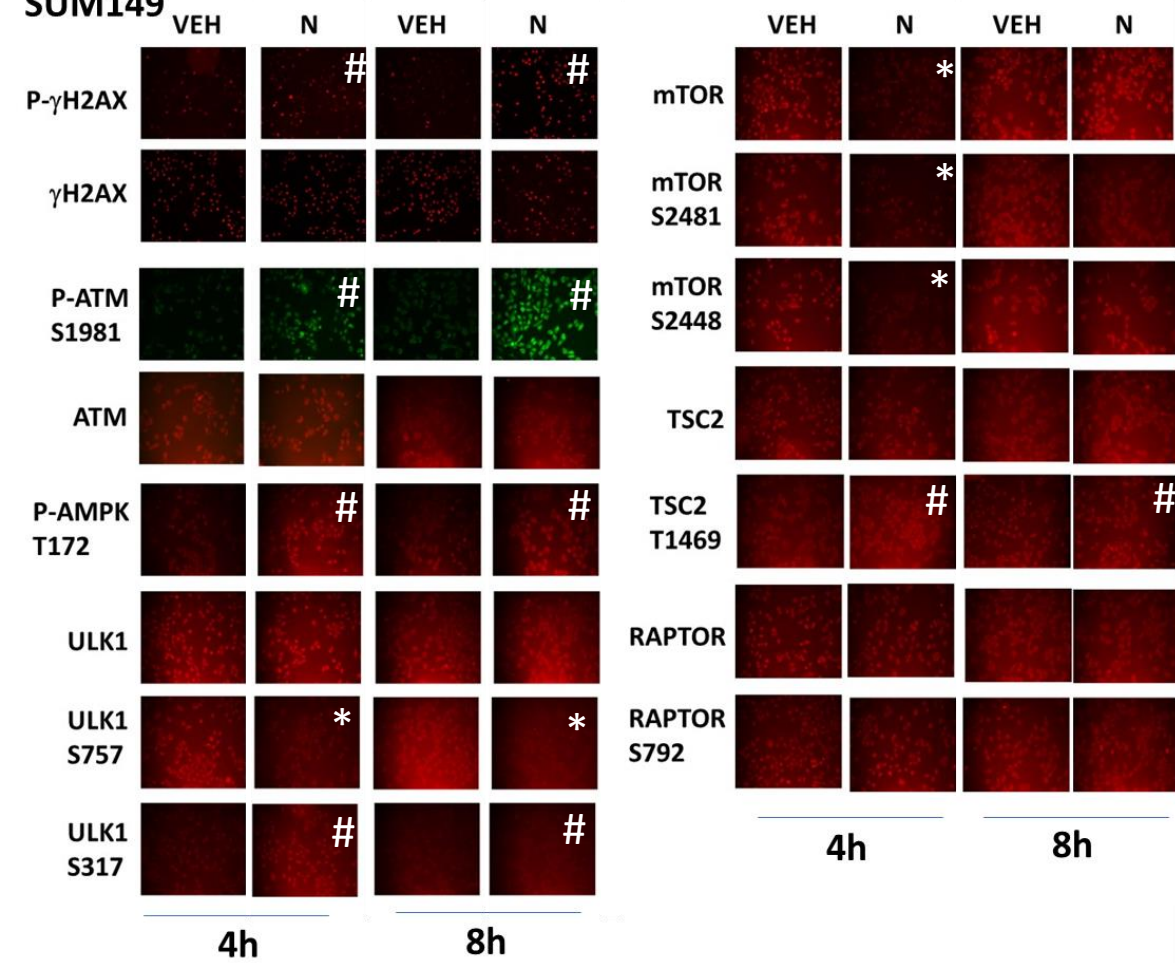
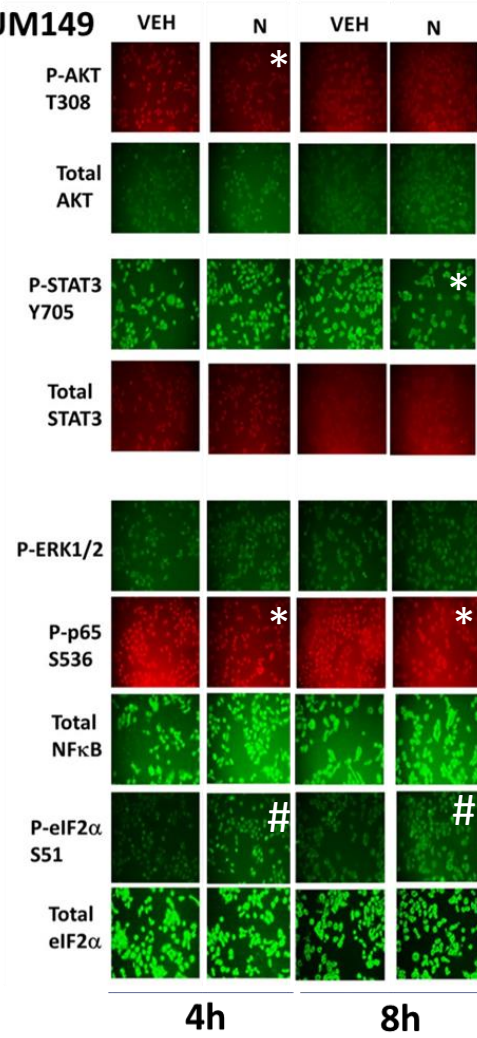


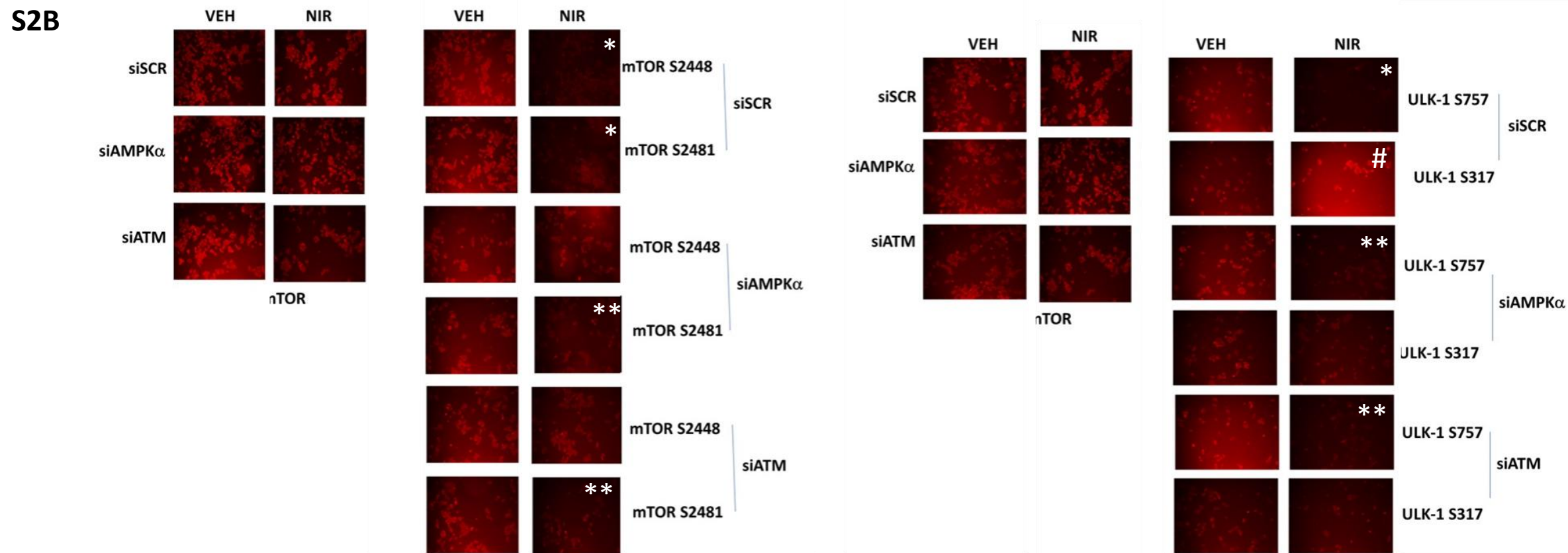
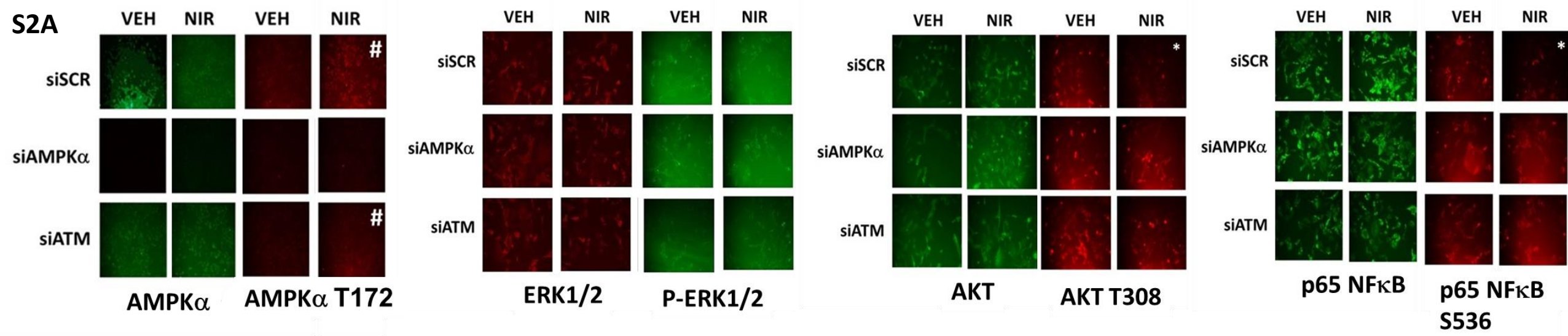
Figure S1. Niraparib activates an ATM-AMPK-ULK1-autophagy pathway in SUM149 TNBC

cells. SUM149 cells were treated with vehicle control or niraparib (2 μ M), for 4h and 8h. Cells were fixed in place and at least forty cells per condition were imaged in independent triplicate and the intensity ratio of phosphorylated protein levels to total protein expression plotted as a percentage of control treatment (n = 3 +/- SD). # p < 0.05 greater than vehicle control; * p < 0.05 less than vehicle control.

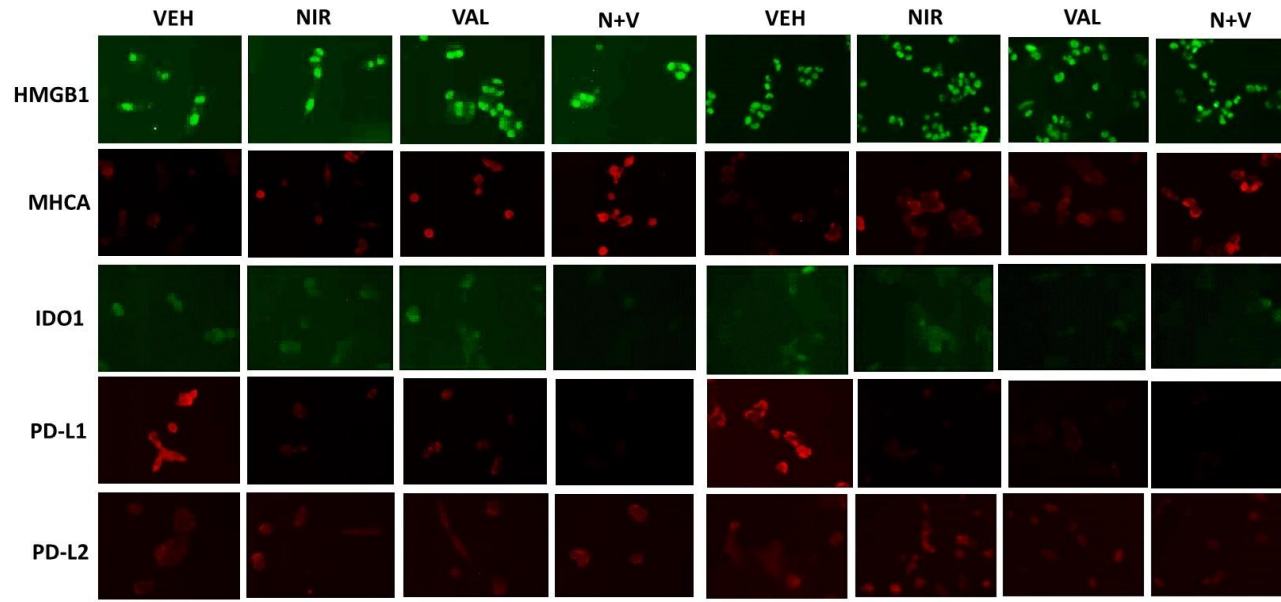
Figure S2. Knock down of ATM or AMPK prevents the dephosphorylation of AKT T308, p65 NF κ B S536, ULK-1 S757, mTOR S2448 and mTOR S2481, and the phosphorylation of ULK-1 S317. A. and B. SUM149 mammary cancer cells were treated with vehicle control or with niraparib (2.0 μ M), for 6h. Cells were fixed in place and at least forty cells per condition were imaged in independent triplicate and the intensity ratio of phosphorylated protein levels to total protein expression plotted as a percentage of control treatment (n = 3 +/- SD). # p < 0.05 greater than vehicle control; * p < 0.05 less than vehicle control; ** p < 0.05 less than vehicle control but greater than corresponding value in siSCR cells.

Figure S3. Niraparib and valproate interact to reduce PD-L1 and IDO-1 expression and enhance MHCA levels. **A.** Spiky and OVCAR3 cells were treated with vehicle control, niraparib (2 μ M), valproate (250 μ M) or the drugs in combination for 6h. Cells were fixed in place and at least forty cells per condition were imaged to determine the staining intensity for each protein (n = 3 +/- SD). # p < 0.05 greater than vehicle control; * p < 0.05 less than vehicle control. **B.** Cells from Panel A, were imaged at 60X magnification and the expression and localization of HMGB1 determined.

S1**SUM149****SUM149**



S3A



S3B

Spiky

OVCAR3

